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Table of Contents

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ORIGINAL ARTICLES— PAGE.
"Diagnosis of Intracranial Tumours", by R. M. DOWNES, C.M.G., M.D., M.S., F.R.A.C.S. 103
"The White Man in the Tropics", by A. GRENFELL PRICE, D.Litt. 106
"Hypotension", by W. E. FISHER, M.B., Ch.M., M.R.C.P. 110
"Experimental Reinnervation of the Paralysed Bladder", by HUGH C. TRUMBLE, M.B., B.S., F.R.C.S., F.R.A.C.S. 118

REVIEWS—
Physiology in the Laboratory 122
Hormones and Internal Secretion 122

NOTES ON BOOKS, CURRENT JOURNALS AND NEW APPLIANCES—
Contraception 122

LEADING ARTICLES—
Abortion and the Use of Pastes 123

CURRENT COMMENT— PAGE.
Thyreotoxic Hypertension 124
Œdema of the Extremities 125

ABSTRACTS FROM CURRENT MEDICAL LITERATURUE—
Dermatology 126
Urology 126

BRITISH MEDICAL ASSOCIATION NEWS—
Scientific 128

CORRESPONDENCE—
Amendments to the Lunacy Act in New South Wales 131

BOOKS RECEIVED 132

DIARY FOR THE MONTH 132

MEDICAL APPOINTMENTS VACANT, ETC. 132

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 132

EDITORIAL NOTICES 132

DIAGNOSIS OF INTRACRANIAL TUMOURS.[1]

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IN the past the custom has been for physicians to make the diagnosis of intracranial tumours and their situation, and then to hand the patients over to the surgeons as pure technicians to expose and to remove the tumours in the areas directed by the physician. Cushing years ago came to the opinion that the surgeon must have at least an equal share in the diagnosis and should himself decide on the site of attack. As it was in the surgery of the duodenum and gall-bladder where the correlation of the clinical picture with the "pathology of the living" seen in detail by the surgeon added so much to diagnostic knowledge, so it is in the surgery of intracranial tumours. Though team work is probably the ideal, there are practical difficulties and it is generally the surgeon alone who can correlate symptoms and pathology. In this respect the career of one of the two greatest French neurological surgeons is interesting. Until about the age of fifty he was a pure neurological physician; since then he has operated on all his own patients, and as he is able to do at least one operation daily, he probably does more than anyone in the world. Whether we do or do not follow Great Britain and the United States in allowing the neurological surgeon to be paramount in diagnosis, a sound knowledge of neurology is of the highest value to the neurological surgeon and it and intracranial physiology are a very necessary part of his training.

The diagnosis of intracranial tumour is incomplete unless we know the location and the

[1] Part of a paper, entitled "Present-Day Intracranial Surgery", read at a meeting of the Victorian Branch of the British Medical Association on March 7, 1934.

pathological nature of the tumour. Then we may anticipate the course of the disease and the more appropriate methods of treatment, and as experience accumulates it will become more easy to diagnose correctly and to plan treatment more confidently. Most of us were taught that the cardinal symptoms of intracranial tumour necessary for diagnosis were headache, vomiting and optic neuritis; these of course are the classical signs of increased intracranial tension, and while all tumours, if they develop far enough, will give rise to increase of tension, yet we aim to make a diagnosis before this trinity appears, and if possible to prevent by operation their appearance. Many tumours, too, may for a great part of their course fail to cause one or more of these symptoms.

Bailey says that the only cardinal sign of tumour is a steadily increasing afebrile alteration in nervous function. In such case one must exclude or confirm the presence of a tumour, for it is the most serious change of nervous function open to successful treatment. As with other uncommon conditions, the most important step in the diagnosis is for the possibility of an intracranial tumour to occur to the medical man. This will come more frequently with better education of students regarding the frequency and signs of intracranial tumour.

There are several symptoms which should always arouse a suspicion of intracranial tumour. Of these, recurrent vomiting, which is particularly prominent in children, should always put the physician on his guard. It occurs commonly with an absence of nausea and may be projectile in type. It is, however, a very familiar experience for this symptom to be treated for weeks as cyclic vomiting, or even to lead to removal of the appendix, before its significance as a nervous sign is investigated. In adults it is less prominent and more readily suggests cerebral origin.

Repeated headache is a common suggestive symptom at all ages, the more so in adults, since in children the expansion of the skull prior to union of the sutures makes it more transient. It is intermittent, usually present on waking in the morning or on exertion, while the special characteristics of migraine are absent. Enlargement of the head is a children's sign, and, if not due to tumour, is a result of inflammation equally calling for operative treatment. In infants it is likely to be the sole distinct early symptom.

Failure of vision is so often the first danger sign that the diagnosis of tumour frequently comes from the ophthalmologist. When occurring rapidly or before the presbyopic age and especially if associated with recurrent headache, vomiting or mental changes, it is most suspicious of tumour.

So much information is derived from ophthalmoscopy that it must be used in the routine clinical examination. The surgeon should be able to recognize the normal and abnormal appearance of the fundus of the eye, and to distinguish papillœdema, optic atrophy and evidences of arteriosclerosis. Too great stress cannot be laid on the necessity for plotting the fields of vision in every suspected case of tumour, and the surgeon should be able to do this. A Bjerrum screen is sufficient in most cases, with the addition of a simple perimeter such as Traquair's in others. Tumours of the chiasmal region, temporal and occipital lobes are those in which the diagnosis depends most on departures from the normal fields of vision and their changes on repeated examination.

Epileptiform seizures, general or focal, are frequently the first recognized symptoms of tumour, or may be supplementary to changed mental or personal characteristics. Epilepsy is a specially suggestive sign when it first appears in middle life, for very few idiopathic cases begin after the age of forty, more than half becoming evident before the age of fifteen. The main differential diagnosis in such cases is from arteriosclerosis and cerebral syphilis. There is an increasing tendency to associate idiopathic epilepsy with previous injury or infection, sometimes at birth or even during intrauterine life, which has produced a predisposition to epilepsy. Some fresh insult to the brain may reveal this predisposition, and, if in middle age, that insult may be tumour.

A case may be quoted here of a girl of fourteen years who had a history of epilepsy of ten years' duration, beginning as *petit mal*, then appearing as *grand mal* and becoming Jacksonian in type two and a half years ago, with two periods of *status epilepticus*. Six years ago she had a sudden right hemiparesis which partially cleared up. Five months before her admission to hospital headache and vomiting appeared for the first time. On admission she had a right spastic hemiplegia, mental impairment, papillœdema and secondary optic atrophy. Yet her diagnosis still remained encephalitis with thrombosis. She had a large frontal tumour—a cystic astrocytoma.

Repeated cerebral insults such as attacks of giddiness, fainting and transient paresis associated with headache and loss of power of concentration must arouse a suspicion of tumour. Cerebral arteriosclerosis more frequently gives this picture and a differential diagnosis may be impossible by ordinary clinical methods, especially when we know that papillœdema is sometimes present in cerebral arteriosclerosis, and absence of general arteriosclerosis does not preclude its presence in the brain.

While there is no great difficulty in an accurate diagnosis of the position of some tumours, there are many in which it is a matter of the greatest difficulty or impossibility, not only because of lack of localizing signs, but because of confusion of the signs arising from local destruction of nervous tissue with those due to interference with nearby portions of the brain by pressure or œdema, and with distant signs due to a general increase of intracranial pressure or vascular blockage. Consequently it is important to know the earliest symptoms which may be expected to be due to the local dysfunction caused by the tumour.

But frequently this order of symptoms is not found, especially when the tumour is in a silent area. A common example is a cerebellar tumour in

which the first signs are often those of hydrocephalus due to blockage of the cerebro-spinal fluid flow.

Certain signs which might be expected to be of localizing value must be discounted in this regard, such as sixth nerve paralysis which is an early sign of increased intracranial pressure; loss of smell sense, also due to increased intracranial pressure or to diseased conditions of the nose, which seem so common nowadays; rigidity and tenderness of the neck, often due to increased pressure; local tenderness of the scalp and position of the headache. Again adiposity and genital dystrophy may be caused not by pituitary affection, but by a cerebellar tumour causing third ventricle dilatation; and, on the contrary, a suprasellar tumour may cause unsteadiness of gait. Or compression of the cerebral peduncle against the tentorium may cause a homolateral paralysis. Experience and a diagnostic instinct, natural or acquired, mean everything in reaching the correct solutions in such problems.

Further difficulties arise from the absence of history or its unreliability owing to the patient's mental condition, and from inability to carry out satisfactory perimetry owing to his youth or mental condition. Sometimes help may be got in such case from the injection of hypertonic saline solution or hyoscine or even from decompression when the mental condition may be sufficiently improved for a fuller examination.

Bailey states that from 80% to 85% of tumours can be diagnosed by ordinary clinical examinations. It is in the remainder of the cases that ventriculography or ventricular estimation is required and is so often extremely valuable.

As ventriculography is not without risk, it should be limited to such cases and is most conveniently employed as an immediate preliminary to operation; if it furnishes evidence of the situation of the tumour, the operation is proceeded with. The surgeon should always be prepared to go on with the operation in the event of the alarming symptoms that sometimes arise from this procedure. It is of most value in tumours of the frontal lobes. It must be remembered that it is only from displacement and alteration of the size and shape of the ventricles that the presence and position of a tumour can be interpreted.

In many cases no help is obtained, nor will it distinguish blockage due to new growth from that due to adhesions in the condition variously known as arachnoiditis, serous meningitis or "pseudotumour". This condition, not very widely recognized, is by no means a rarity. Typically there is primarily a mild meningitis in which the cerebrospinal fluid contains no organisms and few leucocytes; or it may resemble an influenza-like attack or middle ear suppuration. This is followed in a few months by symptoms due to adhesions which may occur anywhere, but whose favourite sites are the posterior fossa, the chiasmal region or the spine; in the first situation they occur either around the fourth ventricle with resulant blockage of its foramina, or further forward, causing a floating up of the cerebellum or a cyst-like collection of fluid and distortion of the medulla. The initial infection may occur unnoticed in infancy or even in fœtal life. It is complicated in some cases by patchy gliosis anywhere in the brain, especially in the region of the aqueduct of Sylvius when it causes blockage or obliteration of this channel and intense hydrocephalus. In a number of cases where the affection is about the fourth ventricle it is distinctly amenable to operative treatment.

A less well known aid to diagnosis in difficult cases is the method of angiography, originated by Moniz and employed particularly in Portugal and Spain. When one sees Moniz employ it, one realizes how much information it affords regarding tumour location; but experience is very necessary in interpreting the skiagrams. There is no great difficulty in preparing the arteriogram. The common carotid is exposed and an opaque solution is injected into it by a needle. Various opaque substances are used such as 25% sodium iodide solution, chemically pure to avoid unpleasant reactions; but the most satisfactory is a thorium oxide solution known as "Thorotrast". Ten to twelve cubic centimetres are used, but up to 20 cubic centimetres can be injected without ill effect and the arteries on both sides may be injected at the one sitting. An essential point to notice is that the skiagram must be taken immediately the injection is made, as the fluid has passed through and left the cerebral arteries in two seconds. Moniz employs an apparatus that takes six photographs at intervals of a second, but this is not essential. The position of any tumour is deduced from the alteration from the normal position of the various groups of vessels arising from the anterior and middle cerebral arteries, but this requires considerable practice. From Moniz's verified cases it appears that large tumours are most readily diagnosable by this method.

In addition, aneurysms and arteriosclerosis may be recognized; and in some cases the nature of tumours such as arteriovenous aneurysms, meningiomata or astrocytomata may be recognized by the nature of their arterial supply. It is possible that in the future this method may replace ventriculography, but up to the present it has not been sufficiently widely used for its comparative value to be judged.

Apart from these special uses of X ray examination, no investigation of tumour or possible tumour is complete without ordinary X ray examination. By it we may obtain evidence of increased pressure, of alteration of the pituitary fossa, of the presence of tumours that undergo calcification, such as suprasellar cysts, oligodendrogliomata, ependymomata and other slow-growing gliomata and sometimes meningiomata and cholesteatomata. Certain shadows, however, are now and then noted which have no apparent significance such as calcified areas in the falx, calcification of the chorioidal plexuses and of the pineal body. The last sometimes will supply diagnostic information, for displacement of

the pineal shadow from the mid-line shows on which side is the displacing tumour. Thinning or thickening of bone or local erosion may also supply invaluable evidence.

Such a general survey of diagnosis of intracranial tumours leads to consideration of the syndromes of tumours of particular regions of the brain. It should be remembered that the local symptoms are frequently masked by the neighbouring and distant symptoms produced by such tumours.

THE WHITE MAN IN THE TROPICS.[1]

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I HAVE been asked to speak on the very important and unsolved problem of whether the white man, and particularly the Nordic white man, can settle permanently as a worker in the tropics, and I need hardly emphasize how vital is this question to the Commonwealth. Again and again other nations have called us "dogs in the manger" because of our "White Australia Policy" as regards these empty spaces, and only recently the Dean of Canterbury voiced a very usual opinion that we should give North Australia to the Japanese.

But we Australians are only a very small fraction of the white people who are involved in tropical settlement. Britain, France and Holland are profoundly anxious about their great tropical possessions, and I have found American scientists intensely interested in their own tropical problems in Florida, Panama and Puerto Rico, and most anxious to hear Australian views.

The truth is that throughout the world leading scientists are disputing over this question of the ability of white people to settle the tropics. Some Americans, such as the well known Professor Ellsworth Huntington, believe that whites cannot live there permanently because they are destroyed by the climate. Other scientists, like General Gorgas, of Panama fame, and the Australian, Sir R. W. Cilento, consider from the records of Panama and Queensland that the whites can colonize the tropics if they overcome disease. A very great authority, the late Sir Andrew Balfour, took a midway position, but at his death he seemed to be swinging towards Cilento's views.

Let us begin by defining white, settlement and tropics. By white we mean people who are white or nearly white, such as the Europeans, the people of Canada, the United States and Australia, and the near white peoples of Costa Rica or Cuba. By settlers we mean people who live, work and have families for generations in the tropics, and we exclude officials, missionaries, soldiers and traders who go to the tropics only for a time. By tropics we mean the earth's surface roughly between 23·5° latitude north and south, but this covers regions of very varied heat, rainfall and humidity, and some areas will be far more suitable than others for the whites. We can exclude from our study many regions such as the African or Australian deserts, where no one can live, and many countries such as India or Java where the whites will never form a working population as the coloured people and their cheap labour are overwhelmingly competitive. Thus, we can narrow our inquiry to a few possible areas. The most important of these are North Australia, parts of North, Central and South America and the West Indies.

There are three ways in which scientists are attempting to examine the question—the methods of history, of statistics and of the laboratory. Unfortunately history is not a very accurate guide, as the progress of medical science has completely changed white prospects in the last few years. The statistical method is not wholly reliable. It is difficult to secure absolutely satisfactory figures of climate, heredity *et cetera*, even in civilized countries. The laboratory method is also uncertain, for, when you test people under artificial conditions of heat or moisture in a laboratory, you cannot reproduce the exact conditions which face them when they have to undergo acclimatization in a tropical zone. By combining the three methods, however, one can find out a great deal about whether the whites are really making progress.

The history of the white man in the tropics is very fascinating. From the year A.D. 1500 onwards European nations carried out a great pre-scientific conquest of the tropics. The Portuguese, Spaniards, English, Dutch, French and other nations poured into tropical Asia, Africa, America and Australia and either conquered or destroyed the native coloured peoples. Before very long, however, the tropical diseases and the tropical peoples began to regain their own. In India, Java, Africa or Mexico the whites continued to hold sway as governors or traders, but each generation returned home, or if they remained were absorbed like a river flowing into an ocean. In sparsely inhabited countries, such as the West Indies, the whites destroyed the natives; but, instead of working themselves, they brought in negro slaves, and these negroes increased so rapidly that the white masters were soon absorbed. In India the Portuguese tried the interesting experiment of deliberately breeding a half-caste people, but even this proved impossible and ultimately the half-castes will be absorbed. Nevertheless, this white pre-scientific wave left some interesting flotsam and jetsam, and many fascinating books have been written on the little communities which have survived. Central America and the West Indies are full of such groups of white people, and perhaps we might even call the settlement at Darwin one. In 1932 and 1933 I was lucky enough to examine a number of such communities at Costa Rica in the Central American highlands, where an almost pure Spanish community has survived for 400 years; at Jamaica where a German community came about the same time as the German pioneers of South Australia; and at the little and almost unknown island of Saba

[1] Read at a meeting of the South Australian Branch of the British Medical Association on October 25, 1934.

where an English-Dutch community has kept almost pure white since the days of the English buccaneers and first Dutch planters, a period of 250 years.

Without going into scientific details I will simply say that the evidence shows that white men can live and work for generations in the more favourable tropics, provided that they are protected both from disease and from the presence of coloured races, who are usually unhealthy and who are far more dangerous to the white man than any tropical climate. In Costa Rica one found a white Spanish community—artistic and educated—which had kept pure white because the people had been isolated on the plateaux and because the negro had been excluded until comparatively recent days. This exclusion of the negro is of the utmost importance. Only this week a letter came from an American scientist in Costa Rica to say that the Government so fears the rapid increase of the negro population that it is completely prohibiting negro immigration. It is the same in Saba. There one found a very fair type of pure white English-speaking people, who had always done a great deal of their own hard work, but are now in danger, like the Costa Ricans, of being absorbed by negroes.

After the old pre-scientific invasion of the tropics by the whites had failed, a new and far more promising invasion occurred. From the year 1890 onwards the British and American scientists learned the control of hookworm, yellow fever, malaria and many other tropical diseases, and the worst enemies of the whites were partly subdued. On the Panama Canal, for example, "the pest hole of the world", the Americans showed that it was possible to secure a lower death rate than in the most healthy, cool, temperate countries, and the white death rate today is lower than even that of Australia or New Zealand. I spent some three weeks with American scientists in Panama and saw white Americans who, with very few vacations in the United States, had carried out the hardest physical labour in the workshops for nearly thirty years. I also saw whites of the second generation who were doing the hard work perfectly well.

There is more doubt as to the effects of the tropical climate on white women, but as for the tropics injuring children, an exhaustive examination in 1930 showed that white children in Panama were on the average and right up to the end of their high school days of higher standard than similar white children in the United States. The same thing is going on in Southern Florida, where white fruit-growers are working in a climate that is truly tropical. The same type of evidence is now coming from the white sugar growers on the Queensland coast, who are actually doing work that the Americans think no white man can possibly perform. We could, however, copy much from the Americans at Panama, for their control of disease, hygiene and sanitation, and their methods of housing, clothing and diet are unequalled anywhere in the tropical world. Our figures in Queensland would be even better if we would follow some of their ideas.

We must not, however, be unduly optimistic because of these successes. What is really happening in Florida and Queensland is that the white man, in particularly favourable regions where economic factors are particularly suitable, is beginning to penetrate the margins of the tropical zone. The great American doctor, General Gorgas, who conquered yellow fever in Cuba and Panama, made the mistake of being over-optimistic because of his successes and thought quite wrongly that the whites would be able to colonize any part of the tropics. In reality, we are just beginning to understand that we are facing a huge and complex scientific problem, and that the future progress of white people in the tropics depends on a large number of geographic and economic controls. To take only one example, even a small region like Panama has great local varieties of climate, and there are probably in the climate, as affecting white people, a number of factors that are yet unknown. Similarly, one race is more suited to the tropics than another, and even in a single race there are some individuals who are suited to the tropics and others who can never acclimatize. This point was strongly emphasized by Dr. Sundstroem when working in tropical Australia.

Another vital control is soil. The world is gradually abandoning the old fallacy that almost all tropical soils are fertile. Australia, for example, would have saved a vast wastage in lives and expense had she realized that her Northern Territory soils are some of the poorest anywhere.

Isolation is also important. We are beginning to realize that loneliness and inbreeding have harmed many white communities more than tropical climates, and that small scattered settlements, such as some of those in North Australia, have little chance of meeting with success. Comfort is also of vital importance, particularly for women. One of the greatest hopes for white settlement in the tropics lies in air conditioning the houses. Very soon the white man in the tropics may be able to control the temperature of his dwelling as easily and effectively as the American controls the winter temperature by central heating. Then again there is the importance of social habits and of food and drink; and here the Americans have done very important work by showing the importance of deficiency diseases such as pellagra—a primary cause of the so-called "poor white" problem—and in outlining a whole "geography of diet". Many failures in the tropics, particularly British failures, have been partly due to ridiculous clothing, heavy unsuitable diets and alcoholic excess. Again and again when a young man drank himself to death in the West Indies, his parents at home were charitably informed that he had died of fever, and that good old whipping post, the tropical climate, took the blame. Again, we are beginning to realize how dependent the white peoples of the tropics are on temperate policies and markets. The Americans turned Puerto Rico and Cuba into lands of one crop industry, dependent on the cool temperate sugar

markets. Now the United States of America is refusing to pay a profitable price for sugar, and when the unhappy, starving Cubans explode in riot and revolution, the supposed instability of a tropical people is blamed.

Most important of all, we are beginning to realize that the greatest barrier to white settlement in the tropics is neither climate nor sickness, but the presence of vast masses of coloured peoples who, as we know from the history of the kanakas in Queensland, lower the standard of living, create reservoirs of disease and form the means by which the whites can shirk doing the essential physical work.

From Washington to the equator every American scientist I encountered said: "You Australians are the wisest people on earth with your White Australia Policy", and this dictum rests on indisputable facts. The health of white people in the southern United States suffers appallingly from the presence of millions of negroes, while the West Indies and Central America are steadily going black. Jamaica, for instance, which once had thousands of white settlers, is now coloured to 96%.

It now remains to consider the history and prospects of white settlement in tropical Australia and to deal with the pre-scientific invasion, which failed; the new scientific invasion, which appears to be making some headway in Queensland, and the factors which will determine whether any nation, white or coloured, can settle the north of this great continent.

As the Spanish say: "There are tropics and tropics"; and we cannot begin to understand the problem of our north unless we realize that we keep a variety of tropics in North Australia. While we possess no equatorial lowlands like the Congo or Amazon basins, we have four other kinds of tropics: the tropical trade wind coast of Queensland, the tropical plateaux, the interior deserts of Western Australia, Queensland and the Northern Territory, and the wet-dry region—that great belt of country, with a monsoonal rainfall in summer and a drought in winter, which runs right around the Australian north and north-west coast. Throughout the world the high plateaux, with their cooler climates, are the most suitable parts of the tropics for white settlement, but in Australia, out of 46,000 square miles of tropical plateaux over 2,000 feet in elevation, only 14,000 square miles of Queensland plateaux have rainfall and soils sufficiently good to support many whites. Also we can eliminate completely, from the viewpoint of any close white settlement (except perhaps for a few temporary mining camps), the whole of the desert or arid regions which have less than fifteen inches of rainfall—regions which comprise not less than 700,000 square miles. Thus we need consider only the Queensland coastal margin, backed by its comparatively small plateaux, and the wet-dry belt of monsoonal country running inland from the north and north-west coast. Australians should never forget that these two regions are of entirely different character. The Queensland coast and plateaux are really promising, for they possess patches of excellent soil and a good and well distributed rainfall from the monsoons and south-east trades. The northern and north-western coastlands are entirely different. Most of the soils are poor, leached, and deficient in plant food. During six to eight months the country is almost drought-stricken. In summer much of it is flooded by terrific rains, some of the rivers rising fifty or sixty feet.

I have already explained how, in the days before modern science, the whites invaded the tropics, and how, in almost every region, their penetration failed. From 1824 onwards the whites entered the Australian tropics, partly as squatters and partly as agriculturalists on the Queensland and Northern Territory coasts. From 1824 to 1849 the British planted small stations, such as Port Essington in North Australia, and South Australia founded and maintained the Northern Territory as a dependency from 1868 until 1911, when the Commonwealth took control. From the sixties onwards Australia also saw the development of northern Queensland by pasturing, agriculture and mines.

This tropical invasion took the usual course. The whites believed that it was impossible for them to work in the tropical climate and they imported various coloured races which proved hotbeds for diseases that affected the whites in turn. From 1863 to 1891 Australians brought 46,000 kanakas to Queensland, and from 1874 onwards the Northern Territory permitted the entrance of thousands of Chinese. Few people now realize that in 1876-1877 the Japanese Government emphatically refused an official offer by South Australia for an extensive Japanese settlement in the Northern Territory, including free transport for the first 200 Japanese. Indeed, the Japanese Government stated that it would resist immigration by force.

This influx of coloured people to our continent had the same tragic results as in the West Indies and other parts of the world. We Australians, who had entered into posssession of what might have been a marvellous biological laboratory of continental magnitude and free from the worst kinds of tropical sickness, brought in unhealthy types of coloured people, who riddled the country and its white inhabitants with tropical disease. In the Northern Territory during the seventies white men and Chinese coolies died like flies, while in Queensland the "dreadful eighties" saw a kanaka death rate four times as great as that of the white inhabitants, and a white mortality that became 50% greater than that of any other State. Yet, even in these circumstances, events showed the fundamental difference between Queensland and North Australia. In both regions pasturing and mining made progress, but while in Queensland the whites and kanakas established sugar, cutton, fruit and other tropical industries, in the Northern Territory, with its seasonal rainfall, poor soil, isolation and pests, such as the white ant and rat, the whites and Chinese met with no success. The close of the century saw North Australia stagnant, save for

cattle, mining and pearling, and for a plantation system of agriculture—as usual, unhealthy—established on the Queensland coast.

Yet, while the pre-scientific invasion failed in Australia, as in other countries, a scientific invasion from 1900 onwards has made progress, as is the case in Panama and in southern Florida, which latter is a moderately tropical region very like the Queensland coast. Under the much criticized White Australia Policy the nation deported the kanakas, and by a health campaign against hookworm, leprosy, malaria and other diseases made the health and vital statistics of Queensland as good or better than those of any other State. To the utter astonishment of the scientists of all nations we established a working population of 300,000 white people in north-eastern Queensland, probably the largest population of working Nordics in any part of the tropics. There is, of course, the question of alien Italian penetration in the most northern sugar districts, and it is very significant that in these areas foreigners or naturalized subjects number no less than 43%. The Sugar Committee of 1931 reported, however, that the flow of alien immigration had declined, that the problem was passing through a transitory stage, and that satisfactory communities would be evolved out of the communities in the far north. White Australians of British extraction have shown that they can do all the heavy labour in sugar and other industries, and before the depression they were bringing the costs of sugar production down towards the cost of production in coloured labour countries. In this respect mechanical improvements will be of vast importance. In Florida and Jamaica I saw machinery which will eliminate almost all the hard manual labour in the sugar industry. While, however, this machinery will improve the prospects for white workers, it will spell stark naked tragedy for the wretched coloured peoples whom the white man has forced into one crop industries.

In 1924 a scientific investigation of certain Queensland towns disclosed remarkable phenomena. Contrary to all previous beliefs, white residents, even of the second and third generations, seemed to be healthy and strong. Tropical born women averaged larger families than immigrant women from the cool temperate zone, and the most healthy people were those who did hard manual work. There remain two dangers in Queensland. First, the experiment is very new and we are by no means certain of the continued effects of climate. Sir R. W. Cilento considers that there is beginning to be a very definite type of north Queenslander or tropical-born Australian, who moves slowly and who conserves his muscular heat-producing energy in every possible way, but that this type is not lacking in muscular strength, while his endurance is equal in his own circumstances to that of the temperate dweller in his. Sir George Buchanan, in his great report on the Northern Territory, produced evidence to the effect that white labour there was from 10% to 35% inferior to that in the temperate zone.

The second danger in Queensland is that the white industries are uneconomic in the sense that their costs of production are far above world average and that Australia is being forced to pay an inordinately high price for such products as sugar and bananas to allow the white population that standard of living which is essential if whites are to survive in the tropics. Yet Queenslanders can justly argue that their industries are now no more uneconomic than most of the tariff-propped industries of temperate Australia. As Keynes and other economists confess, the tariff-mad and nationalistic nations are boxing themselves in water-tight economic compartments, and we must all face artificial industries and a lower standard of life.

While the whites have succeeded in tropical Queensland, in the rest of North Australia, save in cattle, they have met with practically no success. It is a matter for sympathy that in the Northern Territory, for example, the figures of deaths, illegitimate births, serious crime, suicides and drunkenness are far higher than those for any other division of the continent. Such figures are not necessarily due to the tropical climate, for isolation, a mixed population, poor diet and frontier conditions take their toll from the unfortunate people. Nevertheless, it is significant that South Australia and the Commonwealth fruitlessly expended enormous sums of money in vain efforts to develop a huge area which now contains less than 4,000 whites. From 1911 to 1930 the Federal Government spent over £11,000,000 and in 1928-1929 alone made a loss of £576,000, or about £150 per white person, while under Federal control the costs of working the northern and central railways have exceeded the revenue by nearly 100%. It is small wonder that American scientists in a recent world survey have pointed out the utter futility of Australia wasting vast sums in attempting to develop agriculture and close settlement in her north coast lands of poor soil and uncertain monsoonal rains. The only real hope lies in the cattle country which runs across the continent from Queensland to Western Australia, between the central deserts and the coastal regions. Here on stock routes and water supplies we should spend as much as we possibly can, and it is splendid to know that the motor transport unit which the Federal Government is subsidizing may solve the railway problem and is already reducing costs by 50%.

Outstanding questions of North Australia are the aboriginal and half-caste problems. It is now generally recognized that we should try to segregate the blacks, where it is possible, for example, in Melville Island and Arnheim Land. As regards the thousands of aboriginals who have access to settled country, the best we can hope is to absorb them, as the Americans have absorbed the Red Indians. It is interesting to note that a recent Vice-President of the United States was legally an Indian, a ward of the State. The Australian half-castes are now increasing at the rate of 800 *per*

annum, and I believe that the Protector at Darwin is right in trying to marry these half-castes to one another, and the surplus girls to white people, rather than to force them back to the aboriginal camps.

In conclusion, I would say that history and science provide the answer to those who ignorantly criticize our empty north and the policy of White Australia. The only parts of our tropics which any nation, white or coloured, can hope to settle closely are the coasts and highlands of eastern Queensland; and here we have already planted successful white industries and a white population which is apparently teaching the most extraordinary and unexpected lessons to the whole world. The remainder of North Australia is at best a cattle country. We have poured out £17,000,000 in unsuccessful attempts to settle one portion—the Northern Territory. Agriculture with coloured Chinese labour has been an utter failure, and the Japanese very wisely refused our invitations when we invited them in. If, despite such a record, the Dean of Canterbury and Dean Inge and Mr. Beverley Nicholls continue their criticisms of our supposed selfishness, Australians might humbly ask them to visit the West Indies, and to study, as I did recently, the tragic problems of race, health and economics, which were created by the importation of negro slaves.

The practical lesson from all this is that the Australian Governments of all parties should face their problems in the tropics not as questions of politics, but of science, and that before more money is lost in attempts to plant white settlers and tropical industries, they should prepare the way by careful scientific research. To take only one problem, that of agriculture in the Northern Territory, almost every one of the few soil analyses has been disappointing, and even black soil which I brought down from the Adelaide River flood plains proved deficient in potash. Yet, despite the advice of Sir George Buchanan, the Government only a few years back again attempted to foster by subsidy a one crop peanut industry on soils which a later soil analysis proved unsatisfactory.

This is but one of the many ways in which Australia has wasted millions of pounds by attempting to develop her northern tropics without adequate scientific research. One need is at present outstanding. The Americans are considering the establishment at Panama of a research institute which will examine such problems as acclimatization. If, with the cooperation of the Australian Government, this project could be extended to northern Queensland, immensely important results might ensue, for there alone can scientists examine the effects of a tropical climate on a permanent population of working Nordics, unhampered by the presence of coloured people on a lower economic basis, and blessed by a high standard of living and with tropical diseases under adequate control. If such a scientific institute were able to determine the psychological, physiological and other factors which appear to render certain races and individuals more suitable than others for tropical settlement, the world in general, and Australia in particular, would be spared the loss of millions in money and an untold expenditure in ruined hopes and wasted lives.

HYPOTENSION.[1]

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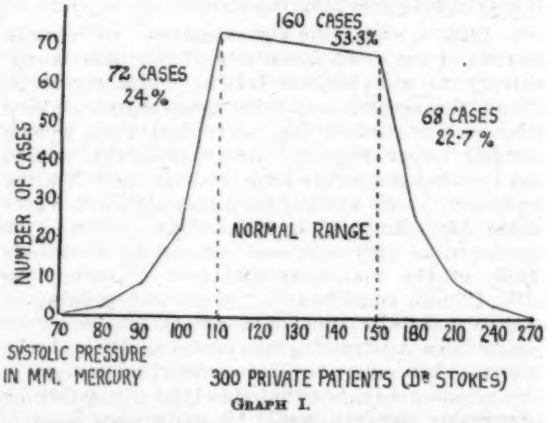
HYPERTENSION is a clinical finding with which we are frequently and considerably concerned, though we still know little of its causation. Hypotension is a condition about which we know less and with which we hardly concern ourselves at all, if one may judge by the scanty references in recent journals and text books. Nevertheless it is quite common. As a preliminary to the writing of this paper I took a series of three hundred consecutive patients in the private consultant practice of Dr. E. H. Stokes. These patients were aged eighteen years and over, but were otherwise unselected. Arranging them according to their brachial systolic blood pressures, it was found that no less than seventy-two, or 24%, had readings of 110 millimetres of mercury or under, and that sixty-eight, or 22·7%, had readings of 150 or over, thus leaving a normal range from 111 to 149 of roughly 50%. Further, it appeared that, if the low and high pressures were arranged in order of severity, that is to say, under 110, under 100, under 90, and over 150, over 180, over 210, and so on, symmetrical curves resulted. Graph I shows these figures arranged in a curve. There has been much difference of opinion



GRAPH I.

among writers as to what constitutes low blood pressure. Standards vary from that of Paul D. White,[1] "below 90 in adults", to 110 chosen by

[1] Read at a meeting of the Section of Medicine of the New South Wales Branch of the British Medical Association on September 6, 1934.

Roberts,[2] both in recent publications. In view of the results outlined above, I decided to adopt 110 as my standard, a decision based entirely on the evidence, as my first impression of Roberts's figure was that it was far too high.

Samson Wright[3] defines blood pressure as "the lateral pressure exerted on the walls of the vessels by the contained blood". Strictly speaking, this must include diastolic and pulse pressures, and intracardiac, arterial, capillary and venous pressures. Of the last mentioned too few figures are available; as regards diastolic, and consequently pulse, pressures, I had to have normal figures for comparison and, these being insurance figures, where the diastolic reading is taken differently from that in ordinary clinical practice, I had to confine myself to ordinary brachial systolic readings. However, since the circulation functions as a whole, consideration of one aspect may give us information as to the behaviour of the remainder.

I have just made the obvious remark that the circulation acts a whole; it should further be remembered that no one part is more important than another, and that it is subject to many and varying external influences. While the possibilities of upset are many, so are the provisions for compensation. For example, the heart muscle itself is influenced not only by its blood supply, but by the degree and direction of control by the central nervous system; the vasomotor mechanism is affected not only by all impulses reaching the centre, including those from the heart, but by its blood supply; and the blood acts not only peripherally by virtue of its physical characters, but universally by its chemical composition, both normal and adventitious.

Simple Examples of Fall in Blood Pressure.

Let us consider now some of the simplest examples of a fall in blood pressure.

1. *Posture.* Normally the blood pressure falls on assuming the erect position. This is rapidly compensated for by constriction of the peripheral bed and an increased heart rate. If the erect position is maintained, motionless, for long, the pressure falls again and syncope may result.

2. *Rest or Sleep.* Here there is a minimum of muscular exertion, the venous return to the right side of the heart is decreased, the left side of the heart fills less, and the heart as a whole beats more slowly, the cardiac output falls and so does the blood pressure. Indeed Hess[4] has recently suggested that we may regard sleep as an example of parasympathetic action in contrast with the classical sympathetic manifestations of emotion and exertion.

3. *Hæmorrhage.* In severe and sudden hæmorrhage the blood volume is rapidly diminished, the venous return and cardiac output are decreased correspondingly, and further, with the loss of hæmoglobin, the oxygen supply to the myocardium and central nervous system is impaired; the heart beats more feebly against a decreased resistance and under a fainter urge. Up to a point tissue fluid movements and vaso-constriction will compensate for these losses; beyond this point, unless we aid Nature by such measures as raising the foot of the bed, bandaging the limbs, intravenous administration of saline solution or blood transfusions, so increasing the available blood volume and oxygen, the malnutrition of the myocardium and of the vasomotor centre may proceed so far as to prevent their compensatory action and the downward trend will go unchecked. I would add here the obvious riders that in a patient who has sustained one hæmorrhage just short of severe symptoms, or in a person whose pressure is already unduly low, a blood loss of extent innocuous in a normal individual will the more easily bring about a dangerous low level.

4. *Secondary Shock.* For practical purposes secondary shock is akin to severe hæmorrhage, if we take it as a local loss of fluid into the tissues. In experimental animals Holt and MacDonald[5] showed that this loss was equivalent on the average to 57% of the animal's total blood volume. The converse can be seen in Mosso's[6] observations that mental stress of any kind caused diminution in the volume of a limb.

5. *Primary Shock.* On the other hand, primary shock is apparently due to a massive effect on the vasomotor centre, with generalized capillary dilatation, and can be compared with the fall of blood pressure seen in the next condition to be mentioned.

6. *Prolonged Ether Anæsthesia.*

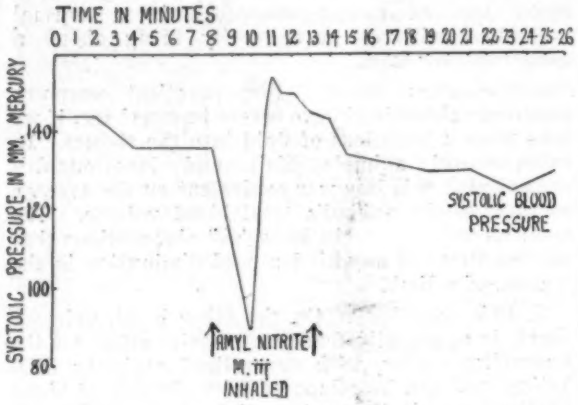
7. *Spinal Anæsthesia.* A similar result, due to a more distal lesion, may be seen in the fall of pressure occurring in spinal anæsthesia.

8. *Amyl Nitrite Administration.* In amyl nitrite administration there is a precipitate fall of blood pressure, followed by an almost equally rapid rise to a higher level than before, and a gradual subsidence to a level a little lower than the original. This is associated with a rise in pulse rate and an increase in cardiac output, which initiate compensation while flushing is still marked. The action of amyl nitrite is directly on the muscle of the vessel walls. The compensation is primarily central and cardiac, with a later peripheral return to normal. I think nothing demonstrates the extreme lability of the blood pressure balance better than such an experiment, of which I show you an illustration (Graph II).

9. *Ventricular Tachycardia.* There are cases of ventricular tachycardia (Lewis[7]) in which the diastolic pause is so brief and the muscular contraction so feeble that the blood pressure falls profoundly.

Now these simple examples have been selected designedly. They have shown you that a fall in blood pressure may be associated with a decrease in blood volume, as in hæmorrhage; with an alteration in peripheral muscular tone, as in amyl nitrite administration; with feeble myocardial action, as in ventricular tachycardia; and with depression of

the vasomotor centre, as in prolonged ether anæsthesia. It is further to be observed that in nearly every instance the nervous side of the mechanism plays an important part, either as the primary cause or as the compensating factor, as, for example, in primary shock and in the reaction to change of posture respectively. Blood volume, peripheral resistance and myocardial efficiency are the classical factors usually considered in the causation of low blood pressure, and we may see their converses in the high blood pressure of erythræmia, hyperpiesia and the secondary cardiac hypertrophy and rise in pressure following on a non-fatal cardiac infarction.


TIME IN MINUTES
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26
SYSTOLIC PRESSURE IN MM. MERCURY
140
120
100
80
SYSTOLIC BLOOD PRESSURE
AMYL NITRITE M. iii INHALED

GRAPH II.

Certain Diseases Associated with Low Blood Pressure.

I pass now to the review of certain diseases in which low blood pressure is considered commonly to occur. (The figures in this group are taken from the records of in-patients in Sydney Hospital.)

1. *Myxœdema.* In the past twelve years I find nineteen cases in which low blood pressure was reported and the diagnosis could be considered definitely established. (Many more patients, of course, have been admitted, but usually only for estimation of the basal metabolic rate, and from these the blood pressure record is lacking.) As you will see in the accompanying table, the basal metabolic rates range from –8% to –39% (Table I). These nineteen patients with an average age of forty-four years had an average basal metabolic rate of –21% and an average systolic pressure of 123 millimetres of mercury. Only four of the patients had systolic readings of 110 or under, namely, 108, 108, 106, 100. All these were women, that is 23·5%, which is a very small increase over the proportion of low pressures among all women in the series I mentioned at the beginning, which was 21%. So while it may seem that the blood pressure in myxœdema tends to be low for the patient's age, definite hypotension below the critical level is not a feature of the disease.

2. *Myasthenia Gravis.* Unfortunately for statistics, *myasthenia gravis* is a rare disease. Since 1923 only three cases with blood pressure readings are available, and in these cases the readings are 140, 120, 112, which does not confirm the suggestion that hypotension is a common feature.

TABLE I.
Myxœdema.

Initials.	Sex.	Age.	Systolic Blood Pressure in Millimetres of Mercury.	Basal Metabolic Rate.
E.G.	F.	45	125	–8%
I.D.	F.	27	120	–20%
J.W.	M.	32	138	–25%
E.G.	F.	25	130	–18%
I.G.	F.	37	112	–15%
A.T.	F.	69	120	–29%
U.B.	F.	27	120	–19%
F.S.	F.	54	140	–9%
J.C.	F.	58	130	–9%
W.S.	M.	62	135	–39%
E.M.	F.	49	120	–22%
H.T.	F.	66	108	–11%
I.H.	F.	37	100	–20%
I.N.	F.	44	120	–10%
N.B.	F.	40	120	–25%
E.E.	F.	47	146	–24%
H.S.	F.	51	106	–32%
P.G.	F.	33	108	–25%
N.S.	F.	27	128	–24%
Average	.. ..	44	123	–21%

3. *Pulmonary Tuberculosis.* I was able to find twenty-one blood pressure readings in the last hundred cases of pulmonary tuberculosis recorded. Of these, eight showed systolic pressures of 110 millimetres or under. If we exclude the lowest two, one of 90 of a patient who also had rheumatic mitral disease and *delirium cordis* at the time of the reading, and one of 96 of a patient who was in an advanced stage of cachexia with secondary carcinomatosis, we are left with six readings of 110, 110, 110, 100, 100, 90, or just under 30%, the three lowest all being readings from men. This is a definite, if slight, increase over the proportion in our series of 300 used as a standard. These also are shown in a table (Table II).

TABLE II.
Pulmonary Tuberculosis.

Initials.	Sex.	Systolic Blood Pressure in Millimetres of Mercury.
C.O.	M.	170
M.C.	F.	160
W.W.	M.	150
P.K.	M.	145
F.D.	F.	145
W.J.	M.	139
P.S.	M.	130
W.C.	M.	130
W.M.	M.	126
L.M.	M.	125
K.M.	F.	120
F.H.	F.	120
H.H.	M.	120
M.B.	F.	110
M.D.	M.	110
M.H.	M.	110
P.S.	M.	100
N.T.	M.	100
A.M.	M.	96
C.S.	M.	90
J.L.	M.	90

4. *Addisonian Anæmia.* In thirty-three available readings in Addisonian anæmia seven cases were of the order of 110 or under, or 21%, no increase on

the incidence in the standard series. Three points of interest, however, emerge from these figures. As you will see from the table (Table III), while only eighteen of the group of thirty-three were males, or 54%, of the seven patients with low readings six were males, or 84%. On the contrary, I would draw your attention in this small table to the fact that six of the seven patients with highest readings are females, a point which we shall meet again later. Further, in every case in which the blood pressure reading was 110 or under, the red cell count was below two million per cubic millimetre, with one exception, in which it was 2,300,000.

TABLE III.

Addisonian Anæmia.

Patient.	Systolic Blood Pressure Reading.
Mary G.	180
Annie L.	180
Alice G.	174
Edith J.	160
Evelyn J.	150
Mary P.	150
William W.	150
Ellen L.	146
James P.	135
Chas. L.	135
James B.	130
John A.	130
George L.	130
Edith B.	130
Mary P.	130
Anna L.	130
John G.	120
Cornelius O.	120
Mary P.	120
Tom S.	120
Edward D.	116
Violet P.	116
William S.	115
William F.	115
Mark H.	115
Fred P...	115
Chas. C.	110[1]
Rupert M.	108[1]
Wm. M.	104[2]
Albert C.	98[1]
William B.	95[1]
Robert K.	90[1]
Margaret S.	80[1]

[1] Red blood cells under 2,000,000 per cubic millimetre.
[2] Red blood cells under 2,300,000 per cubic millimetre.

5. *Cardiac Infarction, due to Coronary Embolism or Thrombosis.* Since 1922 there have been 89 cases filed under the heading of coronary thrombosis, but for my purposes this is inaccurate. Disease of the coronary arteries is common after the age of thirty, as has been shown by *post mortem* and radiographic investigation, and doubtless accounts for many and varied manifestations of myocardial insufficiency. Further, it is granted that progressive coronary changes in structure and, *pari passu*, in myocardial function, associated perhaps with prodromal anginal attacks and with progressive alterations in the *R-T* interval in the electrocardiogram, may occur, just as similar changes may develop in the reverse direction after a non-fatal infarction. The electrocardiogram may return by stages to normal, and the anastomotic coronary circulation improve. Both these changes, however, may be commonly associated with a rising pressure; and they are very different clinically, though related pathologically, from the catastrophe of sudden infarction due to coronary thrombosis or embolism, which was originally associated with the classical Pardee curve and with a great fall in blood pressure. I would suggest that the term coronary occlusion, which means an obliteration, should be reserved for cases of cardiac infarction, and that the other cases should be known more accurately as coronary stenosis. On these lines I have selected from the 89 cases 26 which gave evidence, clinical, pathological or electrocardiographic, of a definite myocardial catastrophe. Of these 26 patients all but four were men, and 14, as you will see from Table IV, or roughly 50%, had blood pressures of 110 or under. Of the 12 with pressure readings of over 110, three, or 25%, died. Of the 14 with readings under 110, eight, or 57%, died. Of the four with readings under 100, four, or 100%, died.

TABLE IV.

Cardiac Infarction.

Patient.	Age.	Systolic Blood Pressure Reading.
Gertrude W.	52	200
Charlotte S.	64	200
Alice S.	54	180
Ada L.	69	178
Lewis N.	53	154
John M.	58	145
Wallace M.	49	134
Lewis W.	59	130[1]
Basil R.	53	120
John G.	76	120[1]
James O.	66	120
George M.	63	112[1]
John B.	53	110
Oscar B.	67	110[1]
William A.	65	110[1]
John S.	56	110[1]
James R.	62	110[1]
William S.	58	108
Abraham R.	65	108
Ernest L.	44	104
William J.	58	100
William B.	66	100
Harold R.	63	95[1]
Herman F.	55	94[1]
Charles G.	58	86[1]
Fred. S.	57	60[1]

[1] Died.

Also there were several cases which illustrated Dr. Ritchie's remark at a recent meeting of this Section, that a progressive fall in blood pressure was of ill omen. These figures suggest the view that the degree of fall is of some prognostic significance as well as its progress. Further, I would ask you to observe in the table that all four women not only had pressure readings of over 110, but were definitely of hypertensive levels.

One of them, Mrs. S., was a patient under my own care. When first seen she had a pressure in the region of 300 millimetres of mercury; subsequently she had a typical attack of cardiac infarction, and when I saw her after admission to hospital her systolic reading was 180. She recovered, and now has a pressure of 250, which appears to be rising steadily.

So, while the low blood pressure of cardiac infarction is often absolute, we must realize that in many cases it is only relative, though of equal diagnostic significance. In cardiac infarction low blood pressure definitely shows itself in an abnormal proportion of cases.

6. *Addison's Disease.* We pass to the condition which I have so far nobly refrained from mentioning—Addison's disease. Here we see hypotension in all its glory. Only four definite cases have been recorded since 1922, and in these the systolic readings were 88, 85, 83, 75 millimetres. The lowered pressure is an outstanding feature of the disease. Nevertheless, recent experience is showing us that, as Addison's disease could not be cured with adrenaline, so it cannot always be cured with cortical extract alone. Cases have been recorded in which a combination of cortical and medullary substances was necessary for success.

Discussion of the Problem.

What light have these half dozen conditions shed on the problem of hypotension?

Myasthenia gravis and myxœdema, it would seem, are not strikingly accompanied by low pressures after all. Pulmonary tuberculosis shows a slight increase in the incidence of low pressure, and a more notable feature is the predominance of men (Table II). As a whole, the pernicious anæmia cases show no increase in incidence, but have a marked preponderance of men at the lower levels. Further, there is some suggestion that very low blood counts (red cells) are associated with low pressures. In cardiac infarction we see a much greater incidence of low pressures than in any of the previous conditions, and in Addison's disease we see hypotension as an almost universal feature. In all these conditions there is the common factor that the low pressure is acquired; that is to say, if we were fortunate enough to have a reading taken prior to the illness, as in the case of Mrs. S. already quoted, we should find a higher figure. With regard to the relative aspect of the low pressure of cardiac infarction, this is an acute condition; but we must remember that there are also cases of hypertensive heart disease which manifest a chronic, progressive low blood pressure of relative degree, which is also of evil omen. The history of such a case is in brief as follows:

H.F., aged sixty-three years, was admitted to hospital on May 26, 1928, with a diagnosis of arteriosclerosis and epistaxis. Previously he had been in good health, except for a similar attack twelve years ago. He had sclerosed radial arteries. His apex beat was situated 12·5 centimetres (five inches) from the mid-line in the sixth intercostal space. His systolic blood pressure was 190 and his diastolic pressure 115 millimetres of mercury.

On December 23, 1930, he was admitted to hospital with a diagnosis of coronary sclerosis. He had suffered from anginal pain for four months and from breathlessness for three weeks. He had œdema of the lungs, liver and legs. His systolic blood pressure was 170 and his diastolic pressure 110 millimetres of mercury.

On June 16, 1931, he was admitted to hospital with a diagnosis of myocarditis. He was complaining of pain, breathlessness, cough and rusty sputum. The urea concentration test gave the following figures: 1·7, 2·2, 3·1, 3·4. His systolic blood pressure was 130 and his diastolic pressure 100 millimetres of mercury.

On August 27, 1931, he had an attack of epistaxis and a mitral systolic murmur was discovered. His systolic blood pressure was 110 and his diastolic pressure 80 millimetres of mercury.

On November 12, 1931, he died of congestive failure.

At the base of all these acquired examples ranging from the acuteness of profuse hæmorrhage and the transience of amyl nitrite to the progressive chronicity of Addison's disease, is some factor incompatible with health. In other words, the acquired hypotension that we have so far considered is a sign found in cardio-vascular examination which occurs in many diseases, which arises in various ways, and the significance of which, as with most single signs, depends on its context rather than on itself. No discussion of hypotension can stop here.

With these examples in mind I propose to examine two series of blood pressure readings, one from the 300 cases in a private practice in Macquarie Street already mentioned, the other from a series of 300 in-patients in public hospitals. In the 72 patients with readings of 110 millimetres of mercury and under in the private series, there were four who were suffering from tuberculosis, one who suffered from rheumatic mitral disease, one who had renal glycosuria and three patients with syphilis of the central nervous system. But there was none of the other conditions we have mentioned. The possible explanations of this position may be three: First, 25% may be the normal proportion of individuals with pressures of 110 or under and the fact that they complained of ill health may be an accident. To settle this question I reviewed the blood pressure readings of 283 successful proponents for life insurance, over eighteen years, first-class lives and otherwise unselected. There were so few females, as you will easily understand, that I confine myself to the 268 males. Of these, only 20, or 7·5%, had pressures of 110 millimetres or under; so that low blood pressure appears three and a half times more common among the ailing than in the healthy. In the matter of degree the difference is just as pronounced. Of the 20 low readings in insurance cases 16 were on the 110 mark, and only four below it. In the private patients, 18 out of 40 were below 110, or 45% as compared with 20%.

The question then remained: Was low pressure most commonly due to some disease not so far considered, or was it a matter of no significance? This can best be answered by a survey of the complaints in these cases. Table V sets them out briefly. There are others and they are all of the same type—subjective manifestations for the most part, associated with great frequency with some septic focus, and not infrequently with an old Neisserian infection. Just as their blood pressure mechanism has failed to rise to usual levels, so their resistance to infection seems lowered, and their mental resistance to their environment is below par. Is it going too far to suggest that the existence of the

TABLE V.

Findings in Cases with Systolic Pressures of 110 Millimetres of Mercury and under. Private Practice Series.

Systolic Pressure Readings.	Findings.
84	General debility. Tonsillitis. Prostatitis. Much worry. Neurasthenia.
106[1]	Axillary neuritis. Later ulnar neuritis. Septic tooth.
100	Giddiness and constipation.
90	Arthritis. Septic teeth.
110[1]	Visceroptosis.
110[1]	Occipital neuralgia. Hysterical type.
104	Neurasthenia. Headache. Clammy hands. Vasomotor instability. Septic teeth.
110[1]	Epileptic.
110	Nervous dyspepsia. Nervous type. Septic tonsils.
80	Nightmares. Chronic double *otitis media*, post-influenzal.
110[1]	Tired. Cannot work. Imperfect adaptation to marriage. Menstruation every two months.
110	Recurrent tonsillitis. Headaches. Overwork. Worry.
110[1]	Nervous +++. Dyspeptic. Visceroptotic. Septic teeth.
110[1]	Nervous. Dyspeptic. Formication. Cold spots. Breakdowns. Disappointed in love.
110	Œsophagismus. Vomits on going out with girl friend.
80	Obesity. Gout. Nervous dyspepsia and nervous. Plays thirty-six holes of golf.
110[1]	Giddiness. Tiredness. Family griefs ++. Tonsils ++.
110	Very nervous type. Psoriasis. Pains all over. Costive. Much worry. Tonsils very septic.
110[1]	Vomiting. Dragging feelings. Obese. Hirsute. Post-panhysterectomy.
110[1]	Septic tonsils. Spondylitis. Backache.
110[1]	Actress. Backache. Neurasthenia. Salpingectomy. Nervous dyspepsia. Irregular menses.
110	Eleven septic teeth. Depressed. Infected frontal sinuses. "Lack of pep."
110	Sinusitis. Anxiety neurosis.
90	Prostatectomy. Cholecystectomy. Septic teeth extracted. Giddy. Nausea. Light headed after changing posture.
110	Duodenal irritability. Diseased appendix and tonsils.
90	Frequency of micturition. Costive. Pains in legs. Extremely neurasthenic.
100[1]	Symptomless. For examination—pus in left antrum.
96[1]	Palpitation. Nervous. Despondent.
102	Peritonitis and fistula at 15. Now 28. Tonsils +. "All of a shake."
100	Neurasthenic type. Sweats easily. Frequent abscessed teeth. Introspective. Son of above 29.
110[1]	Tonsils removed twelve years ago.
100[1]	Always cold. Heart thumps. Faints. Very nervous. Is getting divorced.
110[1]	Nuchal headache. Neisserian. Vaginal discharge. Insomnia.
104	Tonsils and adenoids. Funny pains round heart on exertion. Introspective. Palpitation.
110	Chronically inflamed appendix removed. Vomiting. Lassitude. Hypoacidity. Gastroptosis. Was miserable as a child.
110	Tired. Insomnia. Syphilis at 19. Enlarged prostate. Impotence. Hot flushes.
100	Meningitis in infancy. Fast life. Cannot keep still. Legs ache. Headaches. Worries over trifles. Introspective.
110	Weakness. Headache. Dyspepsia. Unhealthy teeth and tonsils.
110[1]	Retroversion. Septic right tonsil. "Nerves." Tires easily. Backache. Unhappily married.
110	Grossly introspective. Gonorrhœa in 1924. Any work breaks him up. Nasal sepsis.

[1]Female.

low pressure is prior to the establishment of the septic focus, and that the septic absorption then intensifies the condition that gave it shelter? I would ask you to remember that all these histories and examinations were made without any thought of the preparation of this paper. Nevertheless, while these subjects of hypotension make poor adjustments to the emergencies of life, they in consequence do not suffer from the effects of all too frequent adjustment, such as hypertensive heart disease, and the ordinary arteriosclerosis of advancing years is probably to them a boon rather than a hindrance, just as time brings relief to the myopic eye and aggravation to the hypermetropic. They lead miserable lives, but they lead them for a long time. So it was that Halls-Dally[8] wrote: "These persistently low pressures do not tend to shorten the prospect of life . . . . on the other hand, they might be bad cases for disability assurance." I would now remind you that in pernicious anæmia and in coronary thrombosis and in pulmonary tuberculosis the incidence of low pressures was more marked in males than in females. Here I find my figures at variance with Halls-Dally's[8] opinion. This preponderance of males at the lower end of the scale is well seen in the private series.

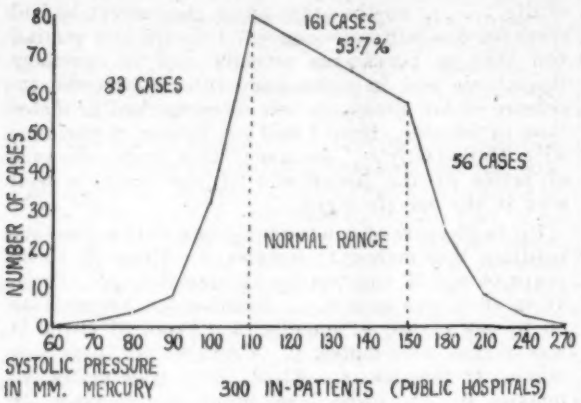
Up to 20 years of age the incidence of low pressure readings was males, 1; females, 3. From 21 to 30 years of age it was males, 11; females, 12. From 31 to 40 it was males, 11; females, 9. From 41 to 50 it was males, 9; females, 3. From 51 to 60 it was males, 4; females, 0. From 61 to 70 it was males, 2; females, 2. From 71 it was males, 2; females, 0. The totals were males, 40; females, 29. If we arrange them in percentages according to severity, of those registering 110 and under, 54% were males; of those registering 100 and under, 82% were males; of those registering 90 and under, 88% were males; of those registering 80 and under, 100% were males.

This is the more interesting when we consider that the hypertensive figures were as follows. Of those registering 150 and over, 59% were females; of those registering 180 and over, 69% were females; of those registering 210 and over, 100% were females. So that males appear to dominate the low readings and females the high, both in frequency and severity.

Insurance figures show us that in addition to the patients who have low pressures associated with some definite disease, and in addition to those in whom the low figures are associated with chronic ill health, there are some persons who apparently have a low pressure and suffer no ill effects whatever. This indeed is the accepted insurance viewpoint, that by itself low pressure means a better life than usual. I was able to get some insurance figures showing the cause of death and the blood pressure at time of proposing. Omitting females, loaded lives and deaths by suicide or accident, I found that out of 93 deaths since 1920, only five persons had pressures of 110 or under, or 5·4%, which is less than the proportion shown above among proponents of 7·5%. In a much larger series of cases, Dr. J. W. Fisher[9] showed that the mortality of proponents accepted with systolic pressures of 100 and under (palpatory method) was only 33% of that expected, as compared with a general mortality rate of 80%.

In order to cast my net more widely, I have examined another series of 300 cases among public hospital in-patients (see Graph III). These figures are summarized briefly in Table VI.

In this series one may naturally expect some variation from the private figures. For example, the patients with low grade morbidity are not likely to become in-patients; patients with high pressure are not often admitted to hospital for that condition *per se*; to some extent we may expect the general level to be reduced following on the



GRAPH III.

reduction in activity. On the other hand, we might expect to find a higher proportion of patients with the low pressure associated with definite disease, and this to some extent proves to be the case. This series shows a higher percentage of low readings

TABLE VI.

Showing Complaints of Public Hospital In-Patients with Pressure Readings of 110 and under.

Age.	Complaint.
Female patients:	
55	Raynaud's disease, neurasthenic.
32	Gynæcological.
40	Fractured humerus.
21	Malaria and abdominal adhesions.
18	Appendicitis—very asthenic.
22	? Cretin.
20	Convalescent bronchopneumonia.
18	Appendicitis.
29	Appendicitis.
26	Six weeks' convalescence gynæcological septic wound.
27	Pleurisy with effusion.
39	Mitral disease
32	Ruptured appendix, wound infected.
22	Appendix.
22	Appendix.
26	Asthenic type. Peritoneal adhesions.
35	Pyelitis.
34	Gynæcological.
48	Mastitis.
44	*Prolapsus ani.*
39	Mitral disease, fibrillation and congestive failure.
68	Fibrillation and congestive failure.
42	Gynæcological.
31	Gynæcological.
31	Extensive burns.
39	Fractured leg.
20	Appendicitis.
23	Appendicitis.
45	Chronic colitis.
35	Septic tonsils and arthritis.
28	? Hydatid of liver.
Male Patients:	
28	Subacute nephritis. Septic tonsils.
23	Appendicitis.
47	Hæmatemesis.
43	Hæmatemesis.
38	Hyperacidity. Spondylitis.
67	? Cerebral tumour.
42	*Diabetes mellitus.*
68	Carcinoma—hæmatemesis.
48	Mitral stenosis. Cerebral embolism.
54	Wassermann reaction +++. Chronic infective arthritis.
54	Coronary occlusion.
33	Mitral disease. Fibrillation. Congestive failure.
66	Malignant cachexia. Severe anæmia.
43	Coronary occlusion.
57	Gastric ulcer.
19	Tonsils—otherwise healthy.
25	Ruptured appendix.
22	Six weeks' convalescence. Severe burns.
28	Extensive septic abrasions.
21	Bronchopneumonia convalescence.
60	Coronary occlusion.

than the first, 28% as compared with 24%, and a slightly lower percentage of high readings, 19% as compared with 22·7%. Nevertheless, it is interesting to note that the group in between remains almost exactly the same (53·7%), thus supporting the view that these upper and lower levels of normal are correctly chosen. The complaints of these patients with low readings fall into the following main groups: (*a*) post-operative, especially gynæcological; (*b*) cardiac disease, especially congestive failure in rheumatic carditis, and coronary occlusion; (*c*) gastric disorders; and (*d*) convalescence from acute infections; but the biggest group of all are still those with chronic sepsis. The other difference in this group is that the sex incidence is approximately equal at each end of the scale: Low readings—males, 40; females, 43. High readings—males, 28; females, 28. The tables will give you an idea of the complaints in individual cases.

So we see in both series that, apart from patients who have low blood pressure as one aspect only of some recognized disease, and apart from those with low pressures found in the course of routine examination, who are apparently healthy (just as we are told the Chinese show pressures 20 to 30 millimetres of mercury below European levels), there remain the bulk of patients who manifest low blood pressure associated with a low grade of chronic ill health. These patients are introspective; they frequently give a history of discomfort on sudden alteration of posture; they show evidence of some septic focus, or give a history of Neisserian infection; and are, in a word, if I dare use it, neurasthenic.

What is the purpose or significance of blood pressure? I would answer that it is a measure of the efficiency of the circulation in maintaining an adequate exchange between the capillary stream and the tissue fluids as regards minute volume, and to some extent in maintaining capillary pressure (which aids filtration). Some of us have had the experience of getting out of bed briskly for the first time after a long and severe illness and promptly fainting. This is partly and usually explained by the view that the vasomotor mechanism is slack and fails to react quickly enough to maintain the cerebral blood supply (the function which was mentioned in our first example). I suggest that there is also the factor that the blood pressure, after a prolonged rest and perhaps toxæmia, is set at a lower level, and that a normal fall reduces it more easily to a symptom-producing state. In other words, we have a predisposing and an exciting factor. This is another way of explaining why we are warned against reducing hypertension unduly. Actually there are conditions of hypertension in which attempts at reduction are safe, and even desirable, as in the excessive spasm of early menopausal high pressure and hyperpietic crises. At the back of the phenomenon of blood pressure throughout the cardiac and arterial and capillary sections of the circulation there is muscular tonus. Tonus implies vital muscular activity and constant

sustaining neuronic impulses; these come from a definite centre which is situated in the floor of the fourth ventricle at the apex of the *calamus scriptorius*. I would remind you of pulse tracings in the physiology department which showed waves of large amplitude; these are the Traube-Hering waves, and they represent rhythmical vasomotor impulses. In many cases I feel strongly that the vasomotor centre is primarily at fault as the result of congenital abnormality, secondarily aggravated by such factors as sepsis and psychic disturbances. Whether this is a negative phenomenon due to interference with vaso-constriction which would justify the term neurasthenia, or a positive one due to over-action of the vaso-dilator mechanism, as suggested in Hess's view of sleep, I hesitate to say. With regard to the latter possibility, it cannot be without significance that many of the hypotensives in the private series improved subjectively, and in some cases their pressures rose after treatment with sedatives, such as "Luminal" and the bromides.

Adrenaline is not normally present in the blood stream (no better proof of this can be found than the recent observation that hypoglycæmia in tumours of the islets of Langerhans is paroxysmal). Its presence is an emergency measure, so it looks as if we must not blame its absence for hypotension generally, just as we now know we must not do so specifically in Addison's disease. Pituitrin, on the other hand, which constricts capillaries in man and which combats shock, is found in the circulating blood of mammals. You know that Cushing has recently indicted the pituitary for the establishment of hypertension. It has been shown in experimental animals that extirpation of this gland causes loss of capillary tone; so I put forward the suggestion that some cases at least of hypotension may be due to pituitary disorder, to which some support may be lent by the fact that in acromegaly[10] the blood pressure is often abnormally low. (The only blood pressure reading in acromegaly which I was able to find was one of 110 in a patient aged fifty-four; it is interesting to note that he also had an old Neisserian infection.)

I believe then that low blood pressure may be divided into two main groups: first, the acquired, in which it is merely a feature of some recognized disease, and, secondly, the congenital. This may be of three degrees: first, mild and uncomplicated, to which the organism can adjust itself well and which is usually symptomless; secondly, of the same mild degree, but complicated by intercurrent changes, chiefly of the nature of sepsis, and producing a fairly characteristic symptom complex. These we may term the essential type of hypotension. The third type I have not yet mentioned. Very few cases have been reported, indeed up to the end of 1927 only five, three by Bradbury and Eggleston[11] and two by Ghrist and Brown,[11] under the title of postural hypotension. These cases show "syncopal attacks on change of posture, with a drop of the systolic blood pressure to shock levels; slow and unchanging pulse rates; lack of sweating; increased distress in hot weather; excess of night over day urine; loss of sexual desire and potency; a false general appearance of youth in comparison with the true age; and pallor of the skin and mucosæ". One cannot but be reminded of Pearson and Wyllie's "lymphatic type",[12] with a pale skin, atonic muscles, slow low tension pulse, low blood pressure and signs of excessive pituitary control. Remember that a pale cyanosed skin means a dilatation of the skin capillaries, which are a vast part of the peripheral bed. It is interesting to note that both of Ghrist and Brown's patients gave a history of previous Neisserian infection, that one had a gastric ulcer radiographically and the other occult blood in the stools. (A recollection of Cushing's neurogenic view of peptic ulcer reminds us once more of the possibility of pituitary influence in hypotension.) I am of opinion that these cases, of which I have not found an example, are due to the action of an exciting factor, such as sepsis on a preexisting essential hypotension, where either the predisposing or the precipitating influence is unduly severe. It should be noted that in Ghrist and Brown's cases symptoms commenced in one patient at the age of twenty-eight years and in the other at thirty-five years, and in both were progressive. Following the nomenclature of high pressures, I think we may fitly term this type malignant hypotension.

Summary.

I have endeavoured to show that blood pressure is a function of the circulation as a whole, and that the factor most frequently concerned in its variation to the lower end of the scale is the vasomotor mechanism. It is further suggested that some part may be played by the pituitary gland.

Figures have been quoted to demonstrate that low blood pressure is by no means an infrequent finding; that 110 millimetres of mercury may be conveniently adopted as a critical level; that males tend to predominate among low readings both in frequency and severity; that while low blood pressure is compatible with good health or may be a feature of recognized disease, these two categories fail to account for the bulk of cases.

Low pressures have been divided into the acquired or symptomatic group in which the low reading is merely one feature of a well recognized condition. Here it may be evanescent, as when amyl nitrite is taken; persistent, as in cachexia; progressive, as in the heart failing after infarction; absolute, as in Addison's disease; or relative, as in the failing heart of high blood pressure; and it may have either diagnostic or prognostic value. Secondly, there is the congenital group: (*a*) those of mild degree, uncomplicated and symptomless; (*b*) those complicated by other diseases, notably sepsis and/or venereal infection, producing a characteristic clinical picture of chronic ill health in the absence of obvious organic disease; and (*c*) rarest of all, cases of marked severity, which represent the reactions of normal blood pressure and of essential hypotension exaggerated to a degree that merits the term malignant.

In conclusion, while this paper from the statistical point of view is based on a small number of figures, it has nevertheless led me to the consideration of blood pressure readings in over 1,400 cases. They are all Sydney figures and all recent. I have endeavoured to be unbiassed and to avoid references as far as possible; indeed, my frame of mind on being asked to read a paper on this subject is well summed up in a phrase of Pasteur's: "I do not know. . . I will investigate." If I have succeeded in showing you that hypotension is more frequent than we think and worthy of more attention than we usually give it, I am well pleased.

Acknowledgements.

My thanks are due to the honorary medical staff of Sydney Hospital for allowing me access to their beds and case records; to several resident medical officers, formerly students at Wesley College, for assisting me in collecting blood pressure readings; for the very great courtesy shown me by the Mutual Life and Citizens' Insurance Company in making their figures available to me; and, above all, to the Secretary of this Section, Dr. E. H. Stokes, who gave me access to his beautifully kept private case-books and assisted me in extracting from them what I needed.

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EXPERIMENTAL REINNERVATION OF THE PARALYSED BLADDER.

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IT is well known that medullated nerves of the involuntary or autonomic nervous system are endowed with powers of regeneration in much the same way as are nerves of the voluntary nervous system. It has been shown in addition that the axones of medullated nerves of the voluntary nervous system will grow down sheaths originally containing axones of the autonomic nervous system and *vice versa* (Langley,[1][2] Cannon, Binger and Fitz,[3] Beattie, Duel and Ballance[4]). The end effect of stimulation of the regenerated nerve fibres is determined, of course, not by the type of fibre contained in the donor nerve, but by the nature of the organ or viscus which the fibres eventually reach. Thus, if the cervical sympathetic trunk be divided and its distal (cranial) segment anastomosed to the proximal segment of the divided hypoglossal nerve or to the vagus nerve, then when regeneration has occurred, stimulation of the donor nerve gives rise to effects resembling in type those observed when the normal trunk is stimulated. The pupil for instance dilates and blood vessels of the skin of the ear contract.

It occurred to the author that it might be possible to reinnervate paralysed viscera, and with this object in view certain investigations have been undertaken, which are described below. The bladder was selected for study as contractions of its walls are easily recorded and the question of reinnervation of the paralysed bladder seemed to be one of some practical interest. Reconstructive surgery in respect of the autonomic nervous system has apparently seldom been attempted. Many of the organs innervated by the autonomic nervous system continue to function more or less efficiently when deprived of their extrinsic nerve supply (for example, the gut and blood vessels). Hence it is that surgeons have been able to divide or otherwise destroy various parts of the sympathetic nervous system without very ill effects, and hence also the probable reason why there has been no insistent call for attempts at reconstruction of damaged parts of the autonomic nervous system.

Methods.

All experiments have been carried out on dogs. At the primary operation, performed under general anæsthesia, one pelvic nerve was divided. In some cases the distal segment of the divided pelvic nerve was anastomosed to the proximal segment of the divided hypogastric nerve of the same side, in others to the proximal segment of the divided obturator nerve. The operation in either case is fairly simple in the dog. In certain of the animals a second operation was performed on the opposite side of the body after the passage of some weeks or months. The animals were then kept for some months. Finally the abdomen was opened and the donor nerve (obturator or hypogastric) was exposed. A piece of string was tied tightly round the urethra a short distance from the bladder and a small rubber tube was introduced into the bladder through a stab incision in the fundus and sutured securely in position. The catheter was connected up by tubes containing water and air to a recording apparatus. Provision was made to vary the resting pressure in the system by means of a reservoir of water which could be raised or lowered as desired. The resting pressure in the system was kept at about six inches of water. A rise in tension of the bladder musculature expelled liquid through the catheter and caused an upward movement of the recording lever.

The appropriate nerves were then stimulated as far from the site of anastomosis as possible by means of a weak faradic current. Precautions were taken to prevent spread of current to other nerves. In order to eliminate reflex effects via the spinal cord, the donor nerve was traced upwards as far as possible and divided. In cases in which the obturator nerve had been used as the donor nerve, it was necessary to divide the distal segment again, that is, distal to the nerve bulb, as invariably some regeneration of nerve fibres passing to the muscles of the thigh had occurred.

Anatomy.

In order that the reader may follow easily the descriptions of operations in this paper, a sketch of the nerves in question, as seen in the dog, is included (Figure I).

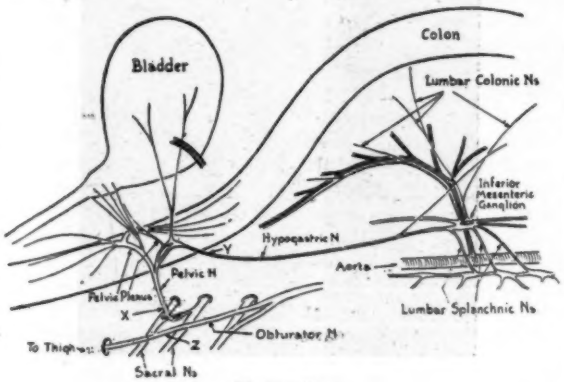

FIGURE I.

Sketch of the nerves mentioned in this paper made from a dissection in a dog. It will be seen that the hypogastric and obturator nerves arise from the spinal cord at a higher level than the sacral nerves giving origin to the pelvic nerves. After dividing the pelvic nerve at the point X, the distal segment may be united to the proximal segment of either the hypogastric nerve divided at Y or the obturator nerve divided at Z.

For a detailed description the reader is referred to another paper by the author.[5]

Experiments.

Dog Number 1. *Bitch.*

The first operation was performed on October 11, 1932. The proximal segment of the divided right hypogastric nerve was anastomosed to the distal segment of the divided right pelvic nerve (double in this case). The animal recovered well, with no obvious ill effects as regards micturition and defæcation. In March, 1933, she produced a litter of pups.

The second operation was performed on May 9, 1933. The proximal segment of the divided left obturator nerve was united to the distal segment of the left pelvic nerve. Recovery was uneventful. After this the animal appeared to have no difficulty in voiding urine. Fæces were passed frequently in small amounts. The adductor muscles of the left thigh were paralysed.

The third operation was performed on October 10, 1933. The abdomen was opened and the right hypogastric nerve was exposed. The left obturator nerve was exposed as high up as possible, well above the site of anastomosis with the left pelvic nerve. The bladder was connected up with a recording lever as described above. Stimulation of the right hypogastric nerve had no effect upon the bladder. Stimulation of the right pelvic plexus caused a powerful contraction of the bladder. This effect is discussed below.

Stimulation of the left obturator nerve, above the site of anastomosis, caused contractions of skeletal muscles in the thigh and contraction of the bladder muscle (Figure II).

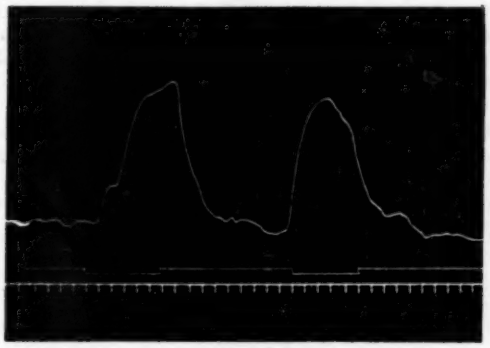

FIGURE II.

Kymographic record of the contractions of the bladder in dog number 1 in response to stimulation of the left obturator nerve, which was anastomosed to the left pelvic nerve five months previously. For details of experiment see text. The upper signal line shows periods of faradic stimulation; the lower records periods of five seconds.

The bladder effect persisted after division of the obturator nerve at its origin from the lumbar plexus and below the site of anastomosis (reunion with the distal segment having taken place), and also after division of the left hypogastric nerve. After the animal was killed the nerves were displayed by careful dissection. On the right side it appeared that part of the pelvic nerve had been left intact at the first operation or had regenerated. Probably this strand was responsible for the positive effect obtained on stimulating the pelvic plexus. On the left side there was

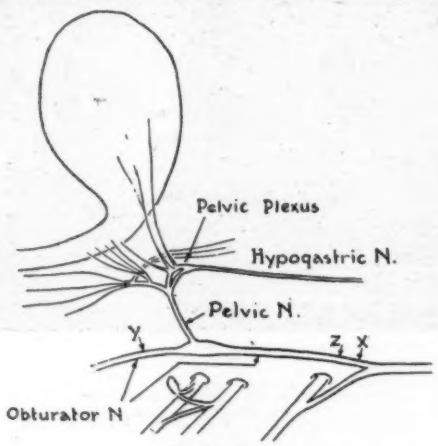

FIGURE III.

Sketch made from a dissection of dog number 1 at the conclusion of the experiment. There is a bulb on the obturator nerve from which the pelvic nerve passes to the pelvic plexus. The distal segment of the obturator nerve had also joined up in the bulb. The origin of the pelvic nerve from the first and second sacral nerves is shown ending in a small terminal nerve bulb. The obturator nerve was divided at points X and Y, after which stimulation at Z gave the effects illustrated in Figure II.

a well formed nerve bulb, in which both segments of the obturator nerve and the distal segment of the pelvic nerve were united (Figure III). The proximal segment of the pelvic nerve was found to end in a small separate nerve

bulb at some distance from the site of union. Histological examination of the distal segment of the pelvic nerve (between the nerve bulb and the pelvic plexus) showed that it contained large numbers of medullated nerve fibres, much larger in size than those normally found in the pelvic nerve.

It seems reasonably certain that fibres grew down from the upper segment of the divided left obturator nerve into the distal segment of the divided pelvic nerve as well as into the distal segment of the obturator nerve. An impulse generated by stimulation of the donor nerve passed along these regenerated fibres to the bladder muscle and caused it to contract strongly. On the right side there was no evidence that impulses generated in the hypogastric nerve reached the bladder.

Dog Number 2. Bitch.

The first operation was performed on November 22, 1932. The hypogastric and pelvic nerves on the right side were divided and the proximal segment of the hypogastric was united to the distal segment of the pelvic nerve.

The second operation was performed on February 21, 1933. A similar operation was performed on the left side. After this operation there was no great difficulty with micturition.

The third operation was performed on May 30, 1933. The hypogastric nerves were exposed and the bladder was connected up with the recording apparatus as usual. Stimulation of the left hypogastric nerve had no effect upon the bladder. Stimulation of the right hypogastric nerve was followed by a definite contraction of the bladder musculature, which resembled in miniature the effect usually seen on stimulation of the pelvic nerve (Figure IV).

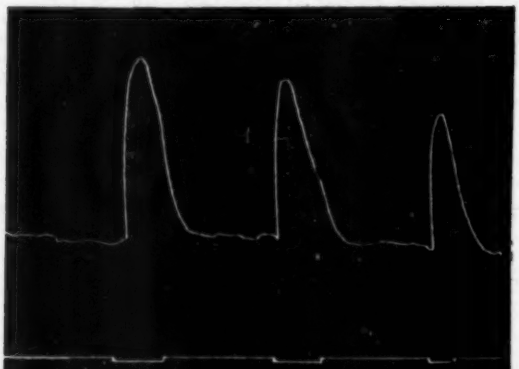

FIGURE IV.

Tracing from dog number 2. Stimulation of the right hypogastric nerve, which had been anastomosed to the right pelvic nerve about six months previously. The time marker records periods of thirty seconds.

The animal was killed. On account of the small size of the nerves and the presence of adhesions, dissection was unsatisfactory.

In this animal regeneration had clearly taken place on the right side. On the left side no effect was noted on stimulation of the hypogastric nerve. The length of the interval allowed for regeneration may not have been sufficient.

Dog Number 3. Bitch.

The first operation was performed on January 31, 1933. The obturator and pelvic nerves on the right side were divided and the proximal segment of the obturator was sutured to the distal segment of the pelvic nerve. About 2·5 centimetres of the right hypogastric nerve were excised.

The second operation was performed on May 16, 1933. A similar operation was performed on the left side, except that the left hypogastric nerve was left intact. After this the animal voided urine without trouble and passed small amounts of fæces at short intervals.

The third operation was performed on October 3, 1933. The obturator nerves were exposed as high above the site of anastomosis as possible. On the left side the nerve was divided well above and below the nerve bulb, on the right side below the nerve bulb. After connecting up the recording apparatus, stimulation of the left obturator nerve above the site of anastomosis was followed by slight but definite contractions of the bladder musculature.

On the right side stimulation caused an effect comparable to that seen on stimulation of a normal pelvic nerve (Figure V).

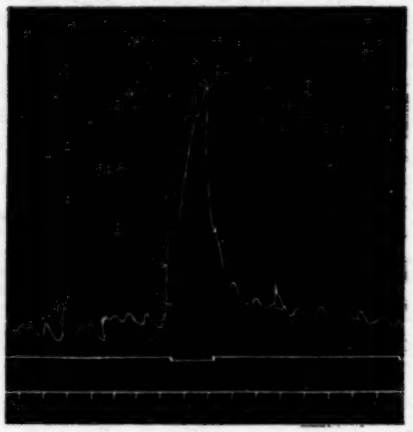

FIGURE V.

Record of bladder contractions on stimulation of the right obturator nerve about eight months after anastomosis with the right pelvic nerve (dog number 3). The time marker records periods of thirty seconds.

It appeared that regeneration had occurred on the left side. The response on the right side was so good as to arouse suspicions that part at least of the pelvic nerve had been missed at the first operation. Dissection, however, failed to demonstrate an intact pelvic nerve.

Dog Number 4. Bitch.

The first operation was performed on November 28, 1933. The right pelvic nerve was anastomosed to the right obturator nerve. The ascending colonic nerves from the left pelvic plexus were divided. This was done for a purpose which need not be discussed here.

The second operation was performed on August 14, 1934. Stimulation of the right obturator nerve caused contractions of muscles in the thigh and also weak but definite contractions of the bladder musculature.

Dog Number 5. Male.

The first operation was performed on November 28, 1933. It was similar to that performed on dog number 4, except that the right vesical vessels were ligated and divided, in order to eliminate the possibility of leaving any accessory pelvic nerve stranda intact.

The second operation was performed on May 22, 1934. Stimulation of the right obturator nerve above the site of the anastomosis was followed by contractions of thigh muscles and fair contractions of the bladder musculature.

Results of Experiments.

Anastomoses were performed in about a dozen animals, but some were liberated, presumably by meddlesome anti-vivisectionists, and escaped. In five of the animals which were subsequently examined for signs of regeneration of nerve fibres, the hypogastric nerve was anastomosed to the pelvic nerve in three instances, the obturator nerve to the pelvic nerve in five instances. The bladder musculature contracted more or less strongly in all cases in which an obturator nerve supplied the regenerating fibres. A positive result was obtained in but one instance in which the hypogastric nerve was used, and the contraction, though well sustained, was not very powerful.

It is evident that in dogs fibres of the obturator and hypogastric nerves may under favourable circumstances grow into the distal segment of the divided pelvic nerve and serve to convey impulses which cause the bladder musculature to contract. The end effect is similar in type to that produced by stimulation of the intact pelvic nerve. The hypogastric nerve contains numerous small medullated preganglionic fibres, whose cell bodies are situated in the main in the upper lumbar segments of the spinal cord, and non-medullated fibres whose cell bodies are to be found mainly in the inferior mesenteric ganglia. There are also a few fibres from other sources which need not be considered. All of the fibres belong to the autonomic nervous system and are not under the control of the will. The number of medullated fibres is far smaller than the number of similar fibres contained in the pelvic nerve.

The obturator nerve belongs, of course, to the voluntary nervous system and contains very numerous medullated fibres of large size, both sensory and motor in type, and also some non-medullated post-ganglionic fibres derived from the sympathetic ganglia via grey rami. Both nerves might be left intact by lesions of the *cauda equina* which would involve the sacral nerves and so cause the fibres of the pelvic nerves to degenerate. Hence, in certain instances, they might be used as donor nerves in an attempt to reinnervate the bladder. The obturator nerve would seem to be the better source of fibres, both because of the abundance of medullated motor fibres and because of the fact that they might be activated voluntarily.

Possible Practical Applications.

Reinnervation of a human bladder, paralysed as a result of some lesion affecting the sacral nerves in the *cauda equina*, would seem to be eminently desirable, but a little consideration will serve to show that the problem is beset with difficulties. In the first place, as regards micturition, in many instances the bladder manages to expel urine at intervals in a fairly satisfactory way, so that no interference is called for. In addition to the visceral involvement there is usually widespread paralysis of the muscles of the lower limbs. The use of an intact nerve, such as the obturator nerve, as a donor will result in the production of additional paralysis of skeletal muscles. In performing the anastomosis very great technical difficulties will be encountered because of the complex nature of the pelvic nerves and plexuses in the human subject, and their intimate relationships with large blood vessels in the pelvis. If in any particular instance these difficulties were overcome and a satisfactory anastomosis between the pelvic and obturator nerves were effected, and if regeneration were satisfactory, it is probable that artificial stimulation of the donor (obturator) nerve would cause the bladder musculature to contract, as in laboratory animals. The subject would no doubt be able, by attempting to put into action those muscles formerly innervated by the donor nerves, voluntarily to initiate impulses which would traverse the regenerated fibres. Thus conceivably the bladder muscles would be placed under the control of the will, and micturition might become a true voluntary act, instead of a more or less involuntary reflex phenomenon. Whether sensory fibres would serve to convey impulses from the bladder to the brain, and what sensations such impulses would evoke, must be left to the imagination of the reader.

It would thus seem that seldom or never will there arise a set of circumstances calling for and justifying an attempt to reinnervate a paralysed bladder in a human being. The circumstances would be somewhat as follows. The lesion would be one which had destroyed the second and third sacral nerves on both sides of the body in such a way as to preclude the possibility of regeneration. There should not be too widespread paralysis of muscles in the lower limbs. Bladder function would be so poor as to necessitate regular catheterization or the institution of suprapubic drainage. The patient should be a good risk and acquainted with the fact that the procedure was quite likely to fail in its object. The surgeon should be a master, skilled in the art of nerve grafting, and with a special knowledge of the anatomy and physiology of the nerves involved.

Summary and Conclusions.

Certain experiments on dogs are described, in the course of which a pelvic nerve was divided and its distal segment anastomosed to the proximal segment of either the divided hypogastric nerve or the obturator nerve. Evidence was obtained that fibres of the donor nerve grew into the distal segment of the pelvic nerve and reached the bladder. Stimulation of the regenerated fibres caused the bladder musculature to contract. In the case of the hypogastric nerve, fibres belong to the sympathetic nervous system, whilst those of the obturator nerve belong mainly to the voluntary nervous system. The pelvic nerve forms part of the parasympathetic system. This effect upon the bladder of stimulation of the regenerated fibres resembled in type that obtained in stimulation of the normal pelvic nerve. The effect was much better and more certain when an obturator nerve was used than when a hypogastric nerve was employed as the donor nerve. Examination of the branches of the pelvic plexus

to the bladder showed that after a successful anastomosis these contained well formed medullated fibres, and it is of some interest to note that when the obturator nerve was used as the donor, the fibres were much larger than those seen normally in the twigs to the bladder. The fact that motor effects were observed in the bladder musculature following upon stimulation of the regenerated nerves, cannot be taken as proof that the new mechanism is capable of subserving satisfactorily the act of micturition. After division of both pelvic nerves, dogs in time usually manage to void urine more or less satisfactorily. Hence the fact that the animals in this series voided efficiently cannot be taken as evidence that the regenerated nerves had anything to do with it. If in a human being the obturator nerve were employed as the donor, the subject would possibly be able voluntarily to initiate contractions of the bladder musculature which would serve to expel urine from the bladder.

The indications and contraindications for attempting some such nerve anastomosis in a human being are discussed.

Acknowledgement.

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Reviews.

PHYSIOLOGY IN THE LABORATORY.

SIR EDWARD SHARPEY SCHAFER is assuredly a *senex paradoxus*. We have here in a fifth edition of his "Experimental Physiology" an up-to-date and very useful guide to practical excercises in the physiological laboratory.[1] Nothing indicates the experienced teacher better than his selection of class practical work, for he admits nothing which has not stood the test of successful usage. The young instructor is too apt to display his erudition by including directions for experiments based on recent discoveries which often fail in the hands of students, and undoubted harm is done both to the student and to the good name of the subject by errors of this nature. The modernity of the book is demonstrated by the inclusion of directions for estimating chronaxie, the Ishihara test for colour confusion and the Lillie wire model of nerve.

Minor criticisms can no doubt be made. For example, the frog's bladder is far superior to either foot-web or mesentery for studying capillary circulation; the spring used in genesis of tetanus could easily be (and usually is) kept in motion by the current it interrupts; more exercises might have been given on special sense, particularly the ear, and in some schools greater emphasis on human physiology will be demanded; but too much attention drawn to these might give the impression that the book is not what it is, a very valuable guide to practical physiology.

[1] "Experimental Physiology", by E. Sharpey Schafer, F.R.S.; Fifth Edition; 1934. London: Longmans, Green and Company. Demy 8vo., pp. 175, with illustrations. Price: 6s. net.

HORMONES AND INTERNAL SECRETION.

THE second edition, improved and enlarged, of Dr. Laquer's book on hormones and internal secretion will be of decided value to investigators and to all who desire a handy work of reference in this field.[1] The scholarship is typically Germanic, but condensation has been carried rather too near to the point at which the volume would become a bibliography rather than a review. That the author is capable of good editing is obvious in those portions where he chooses to exercise his skill, but too often an equality of emphasis makes the book heavy reading. Notwithstanding this defect, we have here the best presentation of the subject yet published. There is a moderately good subject index.

[1] "Hormone und Innere Sekretion", by F. Laquer; 1934. Dresden and Leipzig: Theodor Steinkopff. Medium 8vo., pp. 379. Price: RM 18.

Notes on Books, Current Journals and New Appliances.

CONTRACEPTION.

Two books on contraception have recently been received. The first is "Parenthood: Design or Accident?", by Michael Fielding, with an introduction by H. G. Wells.[2] Why H. G. Wells has been chosen seems rather obscure. On the whole the advice is good, but the Gräfenberg method, involving as it does the introduction of a foreign body into the cavity of the uterus, should not be recommended in any circumstances. We cannot agree that "it should never, on any account, be applied to women who cannot keep in touch with the surgeon". It should not be used at all, surgeon or no surgeon.

The second book, "Birth Control To-Day", by Dr. Marie Stopes, bears the subtitle: "A Practical Handbook for Those who Want to be Their Own Masters in This Vital Matter."[3] It is intended as a book of instruction, shorn of all sentiment. On the Gräfenberg method Dr. Stopes's opinion is sound. She mentions it and similar methods; she states that this can be used only by women under the supervision of a surgeon; but she adds that they should not be used—"they are not only bad, they are so dangerous as to be a positive menace". The methods and teaching of Dr. Stopes are well known, and this book sets them out quite clearly.

Books on contraception are numerous; the subject, of course, is one on which the public is seeking enlightenment. Of the two here mentioned, that by Dr. Stopes is probably the better suited for recommendation by a medical practitioner to his patients. No medical practitioner, however, will recommend to his patients a book that he has not read from cover to cover.

[2] "Parenthood: Design or Accident? A Manual of Birth Control", by M. Fielding, with preface by H. G. Wells; new and revised edition; 1934. London: Williams and Norgate, Limited. Foolscap 8vo., pp. 208. Price: 3s. 6d. net.

[3] "Birth Control To-Day: A Practical Handbook for Those who Want to be Their Own Masters in This Vital Matter", by M. C. Stopes; 1934. London: John Bale, Sons and Danielsson, Limited. Foolscap 8vo., pp. 237, with illustrations. Price: 5s. net.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

ABORTION AND THE USE OF PASTES.

DURING recent years pastes have been used for the induction of abortion. These pastes, the products of continental chemists, were so effective that they were offered for sale to the general public. Therapeutic abortions have occasionally to be undertaken by medical practitioners, and at first sight it would seem that the simple injection of so many cubic centimetres of paste into the uterus would be a very satisfactory way of bringing about the desired result. Though the intrauterine injection of pastes, however, is effective, it is by no means free from danger. After the first wave of enthusiasm for this method had passed, reports of fatalities began to appear, and the literature now contains accounts of many disasters. These are so numerous that the Federal Council of the British Medical Association in Australia has suggested that the matter should be discussed in these pages. Medical practitioners should be warned against using these preparations in their practices; they should also be so well informed that they will be able to warn others of danger. Patients who have made up their minds to terminate an unwanted pregnancy may pay little heed and will sometimes be inclined to submit to injections of paste at the hands of unscrupulous persons. They must be told of the danger of adopting such a course.

In an article that appeared in *Zentralblatt für Gynäkologie,* Welsch pointed out that all pastes used for the production of abortion contained soap and the free fatty acids, and that alkaline solutions had a necrotic action on the epithelium of the ovum and on the uterine wall. He stated that there was no certainty that the corrosive action would not extend to the muscular layers of the uterus. In an autopsy after death from the use of paste, he found loss of epithelium of the uterus and atresia of its cavity. Round cell infiltration of the uterine wall was present, with fresh hæmorrhages into the musculature and considerable pelvic peritonitis. Welsch described a specimen in which extensive necrosis of the entire decidual lining of the uterus was present. He pointed out that as many patients in whom abortion had been produced by paste subsequently required curettage, this decidual necrosis might not be as harmless as one would expect, because of the unknown extent of infiltration of the paste into the uterine wall. He thought that induction of abortion by paste should be given up because of the mortality from embolism and the possibility of the occurrence of necrosis. Sachs, in discussing twenty deaths, stated that the most common cause of death was air or fat embolism. Engelmann reported four deaths from air embolism. Brack reported two deaths. In each instance pulmonary œdema was found *post mortem*; on microscopical examination numerous emboli of paste were found in the smaller blood vessels of the lungs. Another observer carried out animal experiments with one of the better known of the pastes; when death occurred he found that the chief damage was blood cell destruction and injury to the air vesicles and pulmonary tissue. Feldweg reported that at a clinic at Stuttgart several patients had been admitted to hospital with suppurative peritonitis following the use of pastes; one of these patients died. In one case reported

by Janson, despite every care and precaution, severe uterine infection occurred, and this was followed by thrombo-phlebitis of the leg and pulmonary infarction. In another case reported by the same author bilateral parametritis was observed. Seitz reported a case of acute peritonitis that occurred three weeks after the use of a paste. At operation no perforation of the uterine wall was discovered, but it was so sodden and necrotic that infection had spread into the pouch of Douglas and had given rise to the peritonitis. Franken reported the autopsy findings in a case of fat embolism, and expressed the opinion that in the interests of women and of the medical profession the manufacture and sale of pastes of this kind should be prohibited.

Sufficient has been written to show what may be expected to occur after the use of these pastes. The manufacturers of these preparations state that they are simple of application and speedy of action; this is true. They also state that there is no danger associated with their use; this is not true. The scanty references to these preparations in continental literature during the last few months would lead us to suppose that they are not being used as extensively as they were. Should a wider field for their use be sought in Australia, medical practitioners will know how to act.

Current Comment.

THYREOTOXIC HYPERTENSION.

ARTERIAL hypertension has excited great interest in medical circles for some time past, and recently has assumed almost the character of an obsession in the mind of the general public. Thyreotoxicosis is also receiving considerable attention from all aspects, but although the effect of thyreoid hyperfunction on the vascular system has been closely studied, the degree of connexion between it and hypertension has not been given the thought it deserves. Of course it is well known that thyreotoxicosis causes or is associated with a disturbance of the relative levels of the systolic and diastolic blood pressures, for Read's formula for approximate calculation of the basal metabolic rate includes the pulse pressure, but in cases in which the most obvious finding is hypertension the question of a possible thyreotoxicosis is not often considered. John Parkinson and Clifford Hoyle have presented a valuable study of the association of thyreoid toxæmia and essential hypertension in a paper that is notably succinct and clear (a quality not always found in contributions to medical literature).[1] They are careful to point out that they do not call the condition thyreogenic hypertension, for they do not wish to suggest that the hyperthyreoidism is more than one factor in producing hypertension. Wisely they simply use the term "goitre" for the thyreoid enlargement, for a true correspondence between the histological picture in the gland and the observed symptoms cannot be made; however, the effects on the heart and vessels are the same whatever be the exact type of glandular lesion, merely varying with the intensity and duration of the hypersecretion, and probably also with the vascular age of the patient. An extensive literature is summarized. It is evident from this that the association of thyreoid and hypertensive changes has aroused the interest of many writers. More important still is the recognition of masked hyperthyreoidism with hypertension. Parkinson and Hoyle point out particularly that the diagnosis of hyperthyreoidism cannot be unfailingly made by the estimation of the basal metabolic rate, and quote many authors in support of this contention. They further remark that the basal metabolic rate may be normal in true toxic goitre, and is often raised in uncomplicated hypertension. All clinicians who desire to combat the error of making any one laboratory test the touchstone of clinical diagnosis will agree with this. It is therefore important that the diagnosis in the 100 cases studied by these writers should rest on sure foundation, and a consideration of the tables and descriptions in the text of the article assures us of this. Symptoms such as weakness, loss of weight, sweating, tremor, nervousness and exophthalmos have been carefully noted, and the association with the cardio-vascular phenomena, such as tachycardia, auricular fibrillation, high blood pressure, enlargement of the heart, and left ventricular preponderance, has been carefully recorded. The 100 patients thus observed all had established hypertension, in most cases observed over a period of many months. General signs of thyreotoxicosis were found in 86 of them, the commonest symptoms being emotional unrest, nervousness of the type familiar in thyreoid toxæmia, fine tremor, sweating, flushing, wasting and exophthalmos. Thyreoid enlargement was present in all but two patients, and these had previously had part of the gland removed.

Here, then, is a series of cases in which hypertension was easily recognized and in which a chronic goitre was also present, often in masked or easily overlooked form. The most usual type of patient was a woman at or over middle age, thin and excitable; in fact, all but nine of the 100 patients were women. In summarizing their views, Parkinson and Hoyle make this important statement: "In any patient with hypertension showing tachycardia, and still more if there be auricular fibrillation with or without cardiac failure, the thyreoid gland should be examined most critically and supporting evidence

[1] *The Lancet*, October 27, 1934.

of thyreotoxicosis should be sought." The importance of recognizing this conjunction of clinical syndromes will be evident. In treatment it is most essential to know whether the hypertensive patient is also truly hyperthyreoid, for the administration of thyreoid substance is contraindicated, though it is sometimes prescribed, and is the basis of certain proprietary glandular preparations. Moreover, subtotal thyreoidectomy will often relieve the vascular symptoms. Of course it is well known that the slowing down of the metabolic processes in the body will be of benefit to those who suffer from such vascular strain, and in this connexion the recently tried and heroic therapeutic measure of total thyreoidectomy for severe cardiac failure will be recalled. But the subject is really one of importance. On the one hand, arterial hypertension is a condition which probably has no one single cause; it is usually ætiologically vague. Therefore, any case that presents a definite associated factor should be carefully studied, even though we do not go so far as to say that this factor is actually causal. On the other hand, thyreotoxicosis is not always obvious; in its more chronic and masked forms it may elude the unwary physician, who simply does not think of the possibility. Therefore, we should pay serious attention to the careful and guarded conclusion of Parkinson and Hoyle that "the frequency of the combination should revive the suspicion that chronic thyreotoxicosis is one of the causes of hypertension".

ŒDEMA OF THE EXTREMITIES.

IT is well known that the mechanism of œdema in cardiac and renal diseases has been the subject of much research and thought, but the œdema of the extremities is due to several other interesting and often obscure causes. Every practitioner of experience must remember seeing patients, usually women, and often quite young, who complain of one swollen leg. No cause can be found, and no treatment as a rule alters the degree of swelling. This instance, together with the troublesome œdema often associated with inflammation or injury or following operations, serves to point the need for further light on the subject, for œdema, like a skin rash, is a visible and palpable malady, and therefore gives rise to considerable worry to the patient.

Edgar V. Allen has collected data from 300 cases of œdema of the extremities of the lymphatic type, that is, not due to cardiac or renal disease.[1] These cases, studied at the Mayo Clinic during the past ten years, have helped him to present some coherent ideas on classification and causation. He first reviews the anatomy and physiology of the lymph circulation, and certain aspects of this deserve special mention. For example, there is no communication between the superficial and deep systems of lymph vessels in the leg except through the popliteal and inguinal nodes; also all the lymph vessels of the leg join at the groin to pass along the iliac blood vessels. The arrangement in the upper limb is somewhat similar. There is a tendency for the lymphatics to hug the blood vessels closely, a fact that no doubt has an important bearing on certain inflammatory and thrombotic types of œdema. Regeneration of divided lymph channels readily occurs, even in the case of large vessels. One important bearing of the proximity of lymph vessels to blood vessels is seen in "white leg", or *phlegmasia alba dolens*. Allen quotes the experimental work of Homans, who showed that the cause of this condition is obstruction of lymphatic vessels, but that the basic lesion is always in the iliac veins. In classifying lymphœdema, Allen found that nearly one-third of his cases fell into a category for which he uses the qualifying term "præcox". In these the swelling was apparently causeless; it affected only the ankle and foot in some cases, but often extended to the whole limb. His most interesting feature, however, was that 87% of the patients were women, and in 65% of these the onset was during the years of childhood and adolescence, with a special predilection for puberty. The author remarks that the normal limb merely becomes a swollen one; nothing else happens. This remarkable type of œdema has already been referred to; its cause is problematical. The rapid growth of reproductive structures, inadequacy of the venous valves, and possibly some congenital defect in the lymph vessels are cited as possible causes. Other similar cases were congenital, some were familial, and a few both. Those of the familial type belong to the type of chronic hereditary œdema described by Milroy. Besides these primary types there are secondary types due to the occlusion of lymph vessels by metastasis of malignant growths or by fibrous tissue or by external pressure. A form that must be familiar to all is that seen following radical amputation of the breast and associated lymph glands, and occurring apart from metastasis. Allen comments on the remarkable irregularity of the interval between operation and appearance of the œdema. He admits that the cause may be obscure. Metastasis accounts for some cases, and perhaps slight sepsis at operation for others, as Halsted suggested; repeated irradiation seems definitely responsible at times, but why the removal of lymph glands will cause œdema in some instances and not in others still remains obscure. The last group mentioned is that due to inflammatory disturbances, and in this regard attention may be called to the importance of lymphangitis of a chronic nature or recurrent in type. A relatively unimportant variety is that in which œdema of the foot and ankle may be due to a fungus infection of the toes or foot. This might be borne in mind in view of the great frequency of these infections in Australia. The author ends the summary of his article by drawing attention to the shortcomings in our knowledge of œdema of the extremities. Such honesty is necessary. For example, patients with an innocent œdema of one leg have been told it is due to weakness of the heart; this should not happen.

[1] *Archives of Internal Medicine*, October, 1934.

Abstracts from Current Medical Literature.

DERMATOLOGY.

Dermatophytes.

C. W. EMMONS (*Archives of Dermatology and Syphilology*, September, 1934) states that there is a well recognised need in medical mycology for a more logical and usable classification of the dermatophytes, and has attempted this by a natural grouping based on the form of the spores and accessory organs. A small culture cell which is described, was used to supplement plate and tube cultures in studying the development of conidiophores and conidia. The spores are conidia and macroconidia, not aleuries and fuseaux. The conidia vary in size and shape, but these variations may exist within a single species. The conidia of the dermatophytes are characteristic. The manner of spore formation is compared and contrasted with that of certain fungi. The subterminal elements of the branching conidiophore-like structure are really spores, that is, the fungi form short chains of spores. The form of the conidium indicates a close relationship between the different species and genera of the ringworm fungi. The macroconidia are of three types: those of species of trychophyton are clavate and thin walled and have few cross walls; those of lopidermophyton are clavate to egg-shaped and thick walled and have few cross walls; those of microsporum are spindle-shaped and thick walled, and in some species have as many as fifteen cross walls. The type of macroconidium serves to differentiate these three genera, which, as here defined, represent three natural mycologic groups of dermatophytes. The generic names trichophyton and microsporum are retained by right of priority, and epidermophyton (in the sense of Sabouraud) is retained because of established usage. The genera achorion and endodermophyton are unnecessary, their species being distributed between the accepted genera. Chlamydospores and spirals are of little taxonomic value. The "nodular organs" are formed by many species and are probably axogonia. The dermatophytes can be classified mycologically. Such a natural classification follows in a general way Sabouraud's clinical classification. The inconsistencies of that classification can be avoided by shifting the emphasis from the clinical to the mycological aspects. This is not an innovation, but restores the original concepts of the genera. It permits the retention of three familiar generic names. The three natural groups of dermatophytes and the points which characterize them are discussed. The authorship of the genera is enumerated, and the characters of the three genera are set down. A partial list of the synonymy is given. This classification is proposed in the belief that it follows natural lines of relationship and will therefore, if accepted, bring stability to the nomenclature of the dermatophytes. It restores to three ancient genera a mycological definition, which to some degree at least seems to have been the intent of the authors of the names.

Specificity of a Streptococcus Isolated from Patients with Pemphigus.

A. L. WELSH (*Archives of Dermatology and Syphilis*, November, 1934) deals with bacteriological studies of the blood, of material obtained from the naso-pharynx and of fluid from blisters of seven patients who had pemphigus. A streptococcus was isolated from the blood of five of the patients in each of the trials, whereas cultures from the blood of a sixth patient with pemphigus was sterile on four separate occasions. A characteristic streptococcus was isolated from swabbings of the naso-pharynx of these seven patients in each of the twenty-one attempts. A similar streptococcus was obtained from the fluid of blisters only once in thirty-four trials. The streptococcus was found to be virulent for five species of animals, and to produce lesions consistent with pemphigus in a fair number. Intradermal and subcutaneous injections of the dead organism into patients with pemphigus produced bullæ. The different strains have been shown to be serologically identical. Positive precipitin reactions between the naso-pharyngeal washings from patients with pemphigus and the sera of animals immunized to the specific streptococcus are described. The reduction of the mobility of the respective streptococci by the sera from patients with *pemphigus dermatitis herpetiformis, lupus erythematosus* and *erythema multiforme* as compared with their mobility in a solution of sodium chloride is applied as a differential diagnostic test between these four diseases. The streptococcus had a characteristic cataphoretic mobility distribution curve.

Miliary Telangiectasia.

J. E. M. WIGLEY AND G. C. PETHER (*Proceedings of the Royal Society of Medicine*, October, 1934), at a meeting of the Section of Dermatology of the Royal Society of Medicine, reported a case of multiple hæmangiomata associated with changes in the central nervous system. The patient was a male, aged forty years. On the skin and mucous membranes of the lips were numerous hæmangiomata; some were raised and others were flush with the surface, suggesting purpura. Raised lesions were present on the penis, the others were found chiefly round the lower part of the trunk and the upper part of the thighs. The pupils were unequal and reacted sluggishly to light. The tongue and hands were tremulous. The tendon jerks in the arms were brisk. All abdominal reflexes were weak. The lower limbs were moderately spastic; knee jerks were very brisk; clonus was bilateral. The plantar response was extensor. The patient was unsteady on his feet. The authors presented the case for diagnosis. They described the microscopical appearances of a section of skin, and suggested lesions of a corresponding type might be responsible for the changes in the nervous system.

F. Parkes Weber said that similar cases had been described as *hæmangiomatosis miliaris*; he preferred the term miliary telangiectasia because the lesions were telangiectases and not angiomata. He thought that the changes in the nervous system were independent of the skin condition.

Mucha-Habermann Syndrome (Parapsoriasis Varioliformis).

M. SCHOLTZ (*Archives of Dermatology and Syphilis*, November, 1934) describes a condition which is frequently discussed under the name of *pityriasis lichenoides varioliformis acuta*. This condition has often been classed as a parapsoriasis, but the author draws a definite distinction between the Mucha-Hagermann syndrome and parapsoriasis. The morphological and clinical differences are tabulated, and emphasis is laid on the fact that the Mucha-Habermann syndrome clears up comparatively easily, while parapsoriasis does not respond to treatment and runs a very chronic course.

UROLOGY.

Seminal Vesiculography.

A. P. GORRO (*Journal d'Urologie*, March, 1934) considers the various methods of making seminal vesiculograms, and concludes that the best method for routine practice is by vaso-puncture. As a medium, collargol has been found too irritating when used in radio-opaque concentration. The author now uses a 20% solution of neo-iodipin, and finds it very satisfactory. The small bilateral operation is done under local anæsthesia, so that the patient may report when he feels the sensation of fluid entering the posterior urethra, that is, a desire to urinate. Patency of the *vas deferens* is proved early in the injection, and patency of the ejaculatory ducts is proved by the desire to urinate; the latter sensation generally occurs when two to three cubic centimetres of liquid have been injected. The injected liquid remains several days, and sometives even for some weeks, in the seminal vesicles without causing any inconvenience or any functional disturbance. Living spermatazoa have frequently been demonstrated in the seminal fluid a few weeks after vesiculography. The indications for vesiculography are generally con-

nected with chronic inflammatory affections of the vesicles, and in such cases the medium advised by the author has a beneficial therapeutic effect as well.

Chronic Retention in Prostatic Adenoma.

L. MIGLIARDI (*Urologia*, September, 1934) discusses the treatment and prognosis of prostatic adenoma. The consensus of present urological opinion is against suprapubic cystotomy and in favour of slow emptying, followed by continuous drainage through a urethral catheter. The three particular dangers associated with drainage in this condition are hæmorrhage, infection and renal failure. The technique of Colombino, which aimed at prevention of hæmorrhagic and infective complications, is strongly advised. The patient is given an intravenous injection of 10 cubic centimetres of a 40% solution of urotropin and 10 cubic centimetres of a 10% solution of calcium chloride; the former as a prophylactic of infection, the latter of hæmorrhage. A catheter, preferably of the "whistle-tip" type, of size 16 or 17 French is inserted. The outflow of urine is controlled by means of a glass drop apparatus, so that complete emptying takes from six to twelve hours. It is not necessary to use a greater time for emptying; the commonly employed method of allowing several days for emptying possesses the grave disadvantage of favouring infection. The intravenous injection of urotropin should be repeated daily for eight to ten days. The injection of calcium as a rule may be suspended on the second or third day. Even in severe cases the patient's general condition is ameliorated after the third or fourth day. After a week or so a suprapubic drain may be inserted. As for prognosis, on the score of renal efficiency, computation of the alkaline reserve of the blood has been found more reliable than the commonly employed tests, whether of retention or excretion. If the alkaline reserve remains low after some days of catheter drainage, the prognosis is reserved. When the alkaline reserve is below 40 the outlook is gloomy.

Congenital Hypertrophy of the Bladder Neck.

P. CAUSA (*Journal d'Urologie*, August, 1934) draws attention to the clinical entity described by Marion in 1927 as a congenital hypertrophy of the vesical internal sphincter muscle, and describes a personal case presenting a peculiar and remarkable symptomatology. This boy of fifteen passed about seven litres of urine every day and suffered from thirst. On this account he had been treated as a diabetic, though no sugar had actually been determined. A large abdominal tumour was found, and this proved to be a chronically distended bladder containing about two and a half litres of residual urine. In the absence of visually determinable lesions (by X rays, cystoscopy *et cetera*), the diagnosis of congenital hypertrophy of the internal sphincter was made, and this muscle was excised by the suprapubic cystotomy method of Marion, in which a circular incision through the mucosa of the bladder neck is made. At operation the internal meatus was found to be stenosed. Microscopy determined an excessive hypertrophy of the internal sphincter muscle. The plain muscle bundles interlaced freely, running in all directions, the muscle cells were larger than normal, and their nuclei showed increased chromatophilia. Recovery was slow on account of renal damage, but after seven months all vesical residue had disappeared, and the polyuria had decreased by two-thirds; in addition, the general condition was distinctly improved. This disease is somewhat rare, but, on the other hand, it is possibly often not diagnosed. Other types of bladder neck stenosis (not to be confused with true prostatic hypertrophy) have been described by Marion and by Legueu as being due to hypertrophy of local glandular elements (for example, median vesical bar) or of local connective tissue elements (for example, fibrosis resulting from prostatitis), but this entity is a true congenital hypertrophy of the sphincter muscle.

"Shrinkage" Treatment of Prostatic Hypertrophy.

T. J. KIRWIN (*Journal of Urology*, November, 1934) describes the use of the high frequency electric current of a density intermediate between those required for coagulation and for cutting. It is applied transurethrally, the applicator being enclosed in an insulated sheath and having apertures at its distal end through which progress is observed with the McCarthy telescope. Successful treatment depends on the accurate calculation of the current density required to kill epithelial cells without injury to blood and lymphatic vessels. With an electrode of known surface area, the density of current is calculated in "millimils" from the reading of the output meter in milliampères according to the formula:

$$\text{"millimils"} = \frac{\text{milliampères}}{\text{electrode area}}$$

(the electrode area being stated in square millimetres). A "millimil" is defined as a current of one milliampère flowing through an electrode of one square millimetre surface area of contact. The depth of tissue affected by the current depends on the duration of the application and current density. In practice, each area exposed receives from three to six "millimils" for from 45 to 60 seconds. It is calculated that five "millimils" applied to a hypertrophied middle lobe for 45 seconds penetrate to a depth of nine millimetres (three-eighths of one inch). A current of less density applied for a longer time penetrates more deeply. The tissues are blanched, but remain of the original consistency and an indwelling catheter is required for some days. Four case histories are detailed in which treatment was completely successful without any material disturbance of the patient, who were all poor surgical risks. The illustrations of cystoscopic views before, during and after treatment are less convincing, as those of the ultimate appearance of the gland might be readily mistaken for pictures of the pre-operative condition.

Cutaneous Ureterostomy in Vesical Tuberculosis.

J. FRESNAIS (*Journal d'Urologie*, October, 1934) considers that exclusion of the bladder is definitely indicated in rebellious cystitides following nephrectomy for tuberculosis, when (*a*) the intensity of the pain in spite of all treatment renders life intolerable, and (*b*) when the existence of ureteric reflux threatens tuberculous infection of the remaining kidney. Cutaneous ureterostomy in the iliac portion of the anterior abdominal wall gives the best results, and, whatever the final result, the immediate object of relieving the patient of his unbearable vesical pain is always achieved. A rubber catheter is kept permanently in the ureter. It is easily removed by the patient for cleansing and sterilizing purposes and, moreover, the patient can inject mild antiseptics into the kidney whenever required.

Pelvic Cellulitis.

W. S. PUGH (*Urologic and Cutaneous Review*, September, 1934) classifies pelvic cellulitis in the male according to its ætiology as follows: (i) Subsequent to operations (especially electro-surgical) on the prostate gland; (ii) wounds of the lower urinary tract as perforation by cystoscopes or sounds; (iii) due to pericystitis or periureteritis in disease of the bladder or ureter; (iv) venereal diseases with periprostatitis or perivesiculitis; (v) abscesses from other organs affecting the urinary tract by contiguity; (vi) infected diverticula of the bladder or sigmoid colon; (vii) infections from distant foci. Most of Pugh's cases followed transurethral prostatectomy. He believes that pelvic cellulitis due to a blood infection must be very rare, and states that there is enough pathological change present at some time in almost all males to lay the groundwork of a pelvic abscess. The prognosis depends principally on the causation and on the condition of the upper urinary tract. Expeditious treatment improves the outlook. Prophylactic treatment consists in free drainage for all operations on or about the prostate. Perineal section is practised when the condition is active, except when the perineum is extensively scarred. In such cases the posterior vesiculectomy designed by Hunt is used. When there have been previous operations on the bladder or prostate both these organs require drainage.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Medical Society hall, East Melbourne, on March 7, 1934, DR. GERALD WEIGALL, the President, in the chair.

Present-Day Neurological Surgery.

DR. RUPERT M. DOWNES read a paper dealing with present-day neurological surgery. He illustrated his paper by lantern slides and demonstrated a new instrument for craniotomy. The paper was divided into the following sections:

1. Intracranial injuries.
2. Arachnoiditis.
3. Diagnosis of intracranial tumours.
4. Organization and technique of intracranial operations.
5. Surgery of the sympathetic system.
6. Spinal surgery.

The section entitled "Diagnosis of Intracranial Tumours" has been submitted for publication and appears on page 103.

SIR RICHARD STAWELL, in opening the discussion, said that, as a physician speaking on a surgical subject, his best contribution would be to express his appreciation of Dr. Downes's able and interesting exposition, and then to give place to others.

DR. ALAN NEWTON congratulated Dr. Downes on his masterly summary of the present state of affairs in the cranial branch of surgery. The address spoke volumes for Dr. Downes's industry during his recent visit abroad. The essential value of team work in intracranial surgery applied also to other special branches, such as orthopædic, thoracic, goitre and even abdominal surgery. Dr. Newton thought that the reason for his being asked to speak was possibly that about twenty-three years before he had done some research work in this branch of surgery. Since then, however, the elaboration of technique had made it almost impossible to combine this branch with general surgery. The profession in Victoria should welcome the interest of Dr. Downes and Dr. Trumble in Melbourne, of Dr. Lindon in Adelaide and of Professor Harold Dew in Sydney, in this special work. Recent advances had made the picture brighter, and should support Dr. Downes and Dr. Trumble in their efforts. Regarding arteriography, some Japanese surgeons had visited Australia about five years ago, and had given a demonstration of this technique to the Royal Australasian College of Surgeons. They divided the superior thyreoid artery and used the proximal end for introducing the dye. Dr. Newton said that, like most modern workers, he preferred irrigation to swabbing during the course of the operation. With regard to the instruments used in opening the skull, it might be pointed out that the burr at present in use was not a great advance on that used and described by Hippocrates.

DR. J. F. MACKEDDIE congratulated Dr. Downes on his address, and said that he considered that the idea of a young surgeon from Australia going home to serve an apprenticeship to specialists in intracranial surgery was one to be encouraged. Dr. Mackeddie demonstrated some X ray photographs showing the use of insufflation of oxygen into the meningeal space for the relief of indefinite headaches and for post-traumatic symptoms. By this means the cerebro-spinal fluid was replaced by oxygen. He had never been able to demonstrate a block in the meningeal space. The operation was followed by exceeding discomfort, but not by dangerous results, and certain patients had experienced very great relief from chronic severe headache after the resulting discomfort had worn off. This discomfort lasted only about a day, and his patients had said that the relief justified it, and had come back for further treatment. The method used for encephalography was sometimes successful in locating cerebral tumours, but ventriculography was probably preferable.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Anatomy Theatre, University of Adelaide, on October 25, 1934, DR. L. C. E. LINDON, the President, in the chair.

The White Man in the Tropics.

DR. A. GRENFELL PRICE read a paper entitled: "The White Man in the Tropics" (see page 106).

PROFESSOR J. B. CLELAND said that they were all very much indebted to Dr. Grenfell Price for presenting before a medical audience the pros and cons for white settlement of the tropical parts of Australia. Dr. Grenfell Price's interest in the Northern Territory and its history and development had led him to pursue the subject of white colonization in other parts of the world. He had visited America, where there seemed to be exceptional library facilities for acquiring information on the colonization by the Spaniards, Portuguese and others of the tropical parts of America and the East Indies. The information thus obtained he had applied to Australian conditions and he had introduced the matter on numerous occasions in addresses, in print, and over the air to the general public. He had now presented his arguments to a medical audience, for though fertility and geographical position were important factors in the development of any place, the question of the health of the inhabitants was of even greater moment. Could the white man colonize the tropics with success, and, if so, all of them or only part?

In the Northern Territory and adjacent parts, Dr. Grenfell Price had shown that the country in general was remarkably poor and unfit for occupation other than pastoral. In spite of what had been said, it was doubtful whether there was real danger of a foreign power attempting to wrest from Australia such an unpromising area. Nevertheless, there were fertile spots, and an alien establishment in the north would ever be a danger to the southern cities and a ready jumping-off spot for further invasion.

In one sense Australia had been singularly fortunate as regards its tropical portion. There was a native population, but it was in general so sparse and was so unfit to compete with Europeans that it was no menace at all to the white usurpers. Professor Cleland thought that there was no doubt that a few generations would see the extinction of the Australian natives as a pure race, a certain number of half-castes being absorbed into the general population without any detriment to it. These aboriginal people, moreover, had possessed no serious communicable native diseases when the white man first arrived in Australia. Since then, ankylostomiasis, malaria and leprosy had been introduced amongst them, and yaws was also probably a relatively recent introduction. Association with trepang-fishing Malays and introduced kanakas and Australia's proximity to New Guinea were probably responsible for the coming of these diseases, and although they all existed to a moderate extent, they could all probably be readily controlled on account of the scarcity of population. Australians were fortunate, then, in having a tropical part which had no native population of importance, and the few serious tropical diseases that had been introduced could doubtless be readily controlled.

Handicaps to the white occupation of the tropics were several. There was first in most cases a large native population living on a lower social level—a population which did all the hard work, leaving merely the direction to the superior white. Adequate and suitable exercise and occupation were essential to the maintenance of health within the tropics, and with such a subject population the obtaining of these was more difficult. Secondly, the native population had often several diseases readily conveyable to the white population and often affecting them more

seriously than was the case with coloured peoples. Thirdly, alcoholic excess and unwise social habits probably played an important rôle.

Next must be considered the physiological habits of the individual. In hot weather it was an advantage to sweat moderately, as long as the evaporation kept the visible sweat reduced. Some people perspired very slightly and were consequently unfit for living in a warm climate, their temperature inevitably going up with any exercise. Others in a hot dry climate sweated profusely and lost much more water and salt than was necessary. It seemed to Professor Cleland that the suitable settler in the tropics was the one occupying the medium position. There was no doubt at all about the capacity of the white to occupy, generation after generation, the drier type of tropical climate, such as was met with in Central Australia. A race living under these conditions should be thoroughly capable and efficient in every way. Where the conditions were moister (and this was the climate that they usually called tropical), the absence in Australia of an important coloured population and the paucity of diseases communicable from such, reduced the question to whether the individual could adapt himself physiologically to the humid surroundings. Abstinence from alcohol, reasonable exercise and interest, and sweat glands that could adapt themselves to the requirements of the position, as well as suitable clothing, should, he thought, enable the white population to maintain itself, if necessary, for generations under these ideal tropical conditions. Nordic blood, however, probably worked better under the stimulus of adverse climatic surroundings, and the ideal climate for the white man would be one where there were very considerable contrasts between the summer and winter, between day temperature and the night temperature, and between the rainfall and dryness. Personally, he believed there could be hardly any better climate for the development of a virile race of white people than the extratropical climate of South Australia.

DR. F. S. HONE said that before discussing the matter of the paper he would like to congratulate the Scientific Committee for selecting such a speaker and subject. Some members had been feeling lately that with the increase in sections and similar offshoots of the Branch, fresh developments would probably be necessary if the interest in the monthly scientific meetings was to be maintained, and the Scientific Committee had shown vision in making this experiment of a discussion on a subject which was of national as well as medical importance, and of securing Dr. A. Grenfell Price, whose training and recent experiences abroad had eminently fitted him to introduce the subject. Dr. Hone was personally interested in hearing Dr. Price's presentation of the matter, because Dr. Price had shown by his utterances in a paper two or three years ago his dissatisfaction with the administration of the Northern Territory. It was all the more striking that after being with Huntington, who was most antagonistic to the possibility of white settlement in the tropics, and after seeing conditions in America, he should return with such clear-cut convictions as to the necessity of excluding coloured races if white settlements in the tropics were to be made practicable. Dr. Price had given a fascinating address on a most interesting subject. He had set out to show the complexity of the problems and had fully proved his case. By doing this he had rendered members a real service. Much of the talk of white settlement in the tropics was too dogmatic and was inspired either by preconceived notions or political exigencies or similar motives. In his plea for a patient and scientific investigation of the whole matter from different aspects, Dr. Price had taken much higher ground. One rose from reading Huntington's or Griffiths Taylor's writings with a feeling of pessimism as to future possibilities for the white races in the tropics, whereas Cilento was as optimistic in the other direction. One was compelled to agree with Dr. Price that probably the real truth lay between the two extremes. Experience showed that climate alone was not the only determining factor, but, on the other hand, the instancing of small islands like Saba in the West Indies and Kizar in the East Indies to prove successful settlement, proved very little. As Dr. Price had pointed out, conditions in different parts of the tropics were so varied that no conclusion on the whole subject could be drawn from the experiences of any one community. He had dealt fully with the economic side in a very competent and attractive way, but what the members of the Branch were more concerned with were the medical aspects of the question. The profession could show their appreciation of Dr. Price's utterance by attacking these medical problems with fresh vigour. Everyone agreed that housing, diet and clothing were important factors, and the views that Dr. Price had expressed showed how the essentials of shade, ventilation and coolness were being aimed at in the houses in the Panama and in parts of Queensland as well. The reverse was shown in houses in other settlements and in parts of Central Australia. The thing to which as yet not sufficient attention had been paid was the matter of cooling houses, yet as Griffiths Taylor pointed out, the main essential for successful white settlement was the comfort of the labourer's wife. Most attempts at ventilation in the past had been based on the traditions of cold countries, and aimed at providing warm air in the houses. It was a question whether the recent introduction of refrigerating chambers could not be applied on a larger scale to rooms in a house, and whether this would not mark a new era in the solution of the problem of housing for the tropics. On the question of disease there were two things necessary, namely, research into obscure problems and practical application of those measures which research had proved to be necessary. Thinking especially of Australia, one was struck with the way for many years past a number of diseases in north Queensland had been lumped together under such names as "coastal fever". The charts Dr. Price showed illustrated how greatly knowledge of these conditions had advanced in the last few years and how great a variety of diseases were included in that term, and how only this year Weil's disease and an epidemic fever with glandular involvement had been demonstrated to exist in north Queensland. These facts showed the value of combined patient research. At the same time, the recent severe outbreak of malaria in north-west Australia showed the necessity of renewed activity in regard to the measures which they knew would stamp out malaria. Like the economic question, these medical questions were very apt to be governed by political considerations, and Dr. Hone had been wondering whether, as a result of this lecture, the profession could not stimulate activity to tackle these medical problems more definitely from a purely scientific side, as Dr. Price rightly insisted upon. Dr. Hone wished to express his thanks to Dr. Price for a very interesting and instructive address.

A MEETING OF THE SECTION OF MEDICINE OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Robert H. Todd Assembly Hall, the British Medical Association House, Macquarie Street, Sydney, on September 6, 1934, DR. S. A. SMITH in the chair.

Hypotension.

DR. W. E. FISHER read a paper entitled: "Hypotension" (see page 110).

DR. HAROLD RITCHIE said that he had listened to Dr. Fisher's paper with admiration and interest. The paper represented a tremendous amount of work on a subject that was to many a very difficult one. The cases that had always interested Dr. Ritchie were those of essential hypotension—those in which there was no obvious physical cause, or in which no physical cause could be discovered. There was quite a large class of people whose normal blood pressure readings were below the levels cited by Dr. Fisher as normal. There was a large group of people who functioned quite well with a low blood pressure. The next group of cases was that in which psychical causes operated. Dr. Ritchie thought that perhaps in the examination of patients medical practitioners did not always recognize that man was the possessor of two

nervous systems, of which the primitive reacted only through the influences of the emotions. Dr. Ritchie believed that many persons reacted to emotion by a decrease in blood pressure just as others reacted by an increase.

Dr. Ritchie remarked that all medical practitioners were aware that certain diseases, such as cardiac failure, the anæmias *et cetera*, caused hypotension.

Sometimes people with a normally low blood pressure were converted to neurasthenics by the ill advice of their physicians. It was a bad thing to tell the patient what his blood pressure reading was. A person who had been told that he was suffering from low blood pressure would always blame his hypotension for any illness he contracted. An American physician had said: "Many a man enters a physician's room a well man and goes home bowed down under the weight of a column of mercury." In the same way a patient could be made ill by the knowledge of his condition of low blood pressure.

Dr. Ritchie cordially agreed with Dr. Fisher in many of his statements; but he thought that a psychical and a psychological basis should be sought more frequently. He concluded by expressing his thanks to Dr. Fisher for his interesting paper.

DR. JOSEPH COEN extended his congratulations to Dr. Fisher on the vast amount of work that the compilation of the paper must have entailed. The hypotension seen in shock was the outstanding example of low blood pressure, and the dynamic factors were well understood. The lessened cardiac output was the predominant feature, and it was associated with dilatation of the capillaries in the splanchnic area. In hypotension, constriction of the peripheral arterioles was the main factor; it was arguable whether the cardiac output was or was not increased; at any rate, the cardiac hypertrophy was limited to the left ventricle. On the other hand, in hypotension, seen *in excelsis* in shock, a diminution in the cardiac output was the main feature, and the dilatation was not of the arterioles, but the capillaries; this was associated with transfusion of plasma into the tissues and a diminution in the volume of the blood.

Dr. Coen agreed with Dr. Ritchie that the figure, 110 millimetres of mercury, was not low enough for a standard. In a series of cases he should not be inclined to believe that any patient with a systolic blood pressure of 110 millimetres of mercury was suffering from hypotension, providing there was no obvious cause.

Dr. Coen remarked that he was most interested in the type of hypotension due to activity of the vagus. The influence of the emotions on the sympathetic nervous system had also to be borne in mind. Fainting, for example, was an interesting form of hypotension, accompanied by slowing of the heart rate—presumably vagal in origin—relieved by the administration of atropine. He had under his observation a young man who was able to do his daily work of lifting fruit cases into a wagon, but who on a number of occasions, on rising in the morning, had fallen in a faint. This patient had suffered an attack of influenza recently and had not taken sufficient care of himself at that time. His systolic blood pressure on his entry into Dr. Coen's consulting room had been 120 millimetres of mercury; but after he had stood still for ten minutes it had fallen to 80 millimetres, and the patient had experienced similar sensations to those that preceded his fainting fits. He was otherwise a healthy man. Dr. Coen thought that most other types of hypotension were explainable on a dynamic basis.

He concluded by expressing his thanks to Dr. Fisher.

DR. ALLAN WALKER said that he had listened to Dr. Fisher's paper with great interest. He felt that anything he had to say was not based on such a sure foundation as Dr. Fisher's statements; the way to reach sound conclusions was to collect material and to analyse it. He thought that most people had only a vague idea of what hypotension was. If he had to name a figure, it would be below 110 millimetres of mercury; but this was merely arbitrary. Dr. Fisher had pointed out that there were several types of hypotension. First, there was the acquired type; if a patient under observation developed a low blood pressure, a cause had to be sought. The second type was that of the nervous patient, who was neurasthenic and perhaps had chronic sepsis. Many persons with hypertension were also of a nervous type; but the hypertensive person, though nervous, was able to carry on and do his job, whereas the hypotensive was more frequently ineffectual. The influence of the primitive part of the nervous system was important. Persons with hypertension often had paroxysmal hypertension in the early stages, the result of nervous stress; it was probable that persons with hypotension had similar paroxysms. Dr. Walker remarked that he assumed that Dr. Fisher, in speaking of the pituitary gland, meant the whole of the neuroglandular complex.

Dr. Walker went on to say that he always regarded the persons with essential hypotension without symptoms as rather lucky, as they were able to lead an active life without evidence of hypertension or its disadvantages.

With regard to the postural type of hypotension, Dr. Walker remarked that he only remembered one case clearly. The patient was a young girl who had been operated on for appendicitis one month previously; if she stood for any length of time she fainted; in the consulting room her blood pressure fell while she was standing; but the condition was only temporary.

He thought that discussion on such a subject as hypotension did a great deal of good, as it gave some idea of what constituted the normal. Just as people were short, tall, broad or slight, so also they had blood pressures of varying degrees. He agreed with Dr. Ritchie that medical practitioners should not make invalids of people with a column of mercury. The discussion also showed the necessity of taking the blood pressure for the medical practitioner's own information. A change in the blood pressure might be the first evidence noted of the onset of serious disease.

Dr. Walker concluded by remarking that when he had come to the meeting he had not known quite what the term "hypotension" had meant; he felt that the discussion had clarified the situation; he was grateful to Dr. Fisher.

DR. E. H. STOKES congratulated Dr. Fisher on the amount of work he had done. There was just one point on which he wished to comment. Dr. Fisher had remarked that in myxœdema the blood pressure was sometimes high, sometimes low. This was true; but the pulse pressure was practically always low, and this point was one of the important factors in the method of estimating the basal metabolic rate described by Read.

DR. J. H. HALLIDAY was prompted by Dr. Stokes's remarks to ask what relationship there was between any symptomatology ascribed to hypotension as such, and the associated low pulse pressure which was frequently found in such cases. He asked whether Dr. Fisher had anything to say on this matter.

DR. R. S. STEEL said that he wished to ask for advice. He saw a great number of cases of hypotension among allergic people. He had tried various remedies, such as "Luminal", ergot, pituitrin *et cetera*, each time thinking that he had struck a good method, until something happened—there was an emotional disturbance, and down went the blood pressure again, while the patient was subjected to a severe asthmatic paroxysm. He had taken the blood pressure in a series of cases of urticaria. When the patients were free of symptoms the blood pressure was normal; but when they had urticaria the blood pressure fell. There were similar phenomena in asthma. Dr. Steel said that he should be glad to have any information on the means to keep the blood pressure at a normal level in these cases. This measure alone should save these patients from many attacks. He concluded by expressing his thanks to Dr. Fisher for his splendid presentation of the subject.

DR. S. A. SMITH congratulated Dr. Fisher on the excellent manner in which he had attempted a statistical expression of information on the subject of hypotension. Dr. Smith said that he had but vague views of hypotension, and his impressions had not been checked up with statistics. He had been impressed by the number of people who had been made invalids by being told that they had low blood

pressure. He should like to see the Hippocratic oath brought up to date to include the words: "I will not inform the patient of the condition of his blood pressure."

Dr. Smith said that in a vague way he had come to regard blood pressure readings of 100 millimetres of mercury or lower as a matter of course when associated with organic disease; but he was interested in the other types of hypotension. He had often pondered on one patient who had a systolic blood pressure of 90 and diastolic of 60 millimetres of mercury; this patient had represented Australia as a champion athlete and had no symptoms; it was astounding that he should be capable of such vigorous effort.

Dr. Smith went on to say that he had been disappointed in the discussion in that there were a number of people who possessed the power of raising the blood pressure at will who should have been present. He had gathered from his patients that there were many doctors in Sydney who possessed this power. Apparently those people were not at the meeting. In one case he had been told that a course of high living had been prescribed—apparently with satisfactory results.

Dr. Smith concluded by expressing his thanks to Dr. Fisher for the care he had taken in the preparation of his paper.

Dr. Fisher, in reply, said that perhaps he should mention that he had done most of his reading on the subject of hypotension after he had written the paper; he added that in his reading he had found no reason to alter what he had written. He had tried to achieve three objects: first, that his paper should be based on local material; secondly, that it should represent his own views; thirdly, that it should be provocative of discussion. In regard to the third object, he had not encountered half the trouble that he had expected.

In reply to Dr. Steel, Dr. Fisher said that he was not surprised that urticaria, in which there was capillary dilatation, and other allergic phenomena, was associated with hypotension. He had no suggestions with regard to treatment, except to say that ephedrine, given persistently in large doses, had been used successfully in the treatment of malignant hypotension.

In reply to Dr. Halliday, Dr. Fisher said that he wished to apologize for not having discussed the pulse pressure. Though he had records of the pulse pressure, he had left them out, as he had been unable to obtain normal readings for comparison.

Dr. Walker and Dr. Smith had questioned whether the level suggested by him should be regarded as the normal. Dr. Fisher pointed out that his opinion was based on figures, and he was still inclined to believe it correct, as the figures, when plotted graphically, tended to give a perfect curve. In all the more recent articles on the subject "110 millimetres of mercury" was adopted as the lowest normal level. Treadgold, who had carried out extensive investigations, maintained that no adult with a systolic blood pressure below 110 millimetres of mercury was normal.

In reply to Dr. Coen, Dr. Fisher remarked that arteriolar constriction was probably important in hypertension; but the dilatation of the capillaries in hypotension might also be accompanied by arteriolar constriction. In regard to the influence of sepsis, Dr. Fisher remarked that the question resolved itself into the problem: "Which came first, the chicken or the egg?" One had to consider whether the low blood pressure reading or the sepsis was primary. He felt that hypotension occurred too often to be secondary to sepsis.

Dr. Fisher said that he had seen cases illustrating the condition of hypotensive reaction referred to by Dr. Ritchie. In the usual description in the romantic novel, a person turned cold with fright; but some of Dr. Fisher's patients had said that they had become warm; presumably this was due to dilatation of the capillaries.

Dr. Fisher expressed his thanks for the manner in which his paper had been received. He went on to say that as a member of the Committee of the Section, he thought that attention should be paid to the gathering of local material and that, without any personal reference, the younger men should be given the chance to read papers. In regard to the latter view, he mentioned the admirable paper that had been delivered by Dr. Stobo at a former meeting of the Section.

Correspondence.

AMENDMENTS TO THE LUNACY ACT IN NEW SOUTH WALES.

SIR: I have to call attention to a recent amendment of the *Lunacy Act* of 1898, which has had the effect of greatly facilitating admission to a reception house, and I would appreciate it if such amendment could be brought under the notice of the medical profession generally through the columns of THE MEDICAL JOURNAL OF AUSTRALIA.

The section of the amending Act in this regard provides that a person may be received into and detained in a reception house upon the certificate of one medical practitioner in the form of Schedule 2A; a person may also be received into and detained in a reception house on his own written request or on the written request of a relative or friend, if the medical officer of the reception house certifies at the foot of such written request that in his opinion the person proposed for admission is a suitable case for admission to the reception house for observation and treatment.

The form of medical certificate under Schedule 2A is as under:

> I, the undersigned medical practitioner, hereby certify that on the day of 19 , I personally examined (name of person in full) and am of opinion that he is a suitable case for admission to the (name of institution) for observation and treatment.
>
> Dated this day of One thousand nine hundred and .
>
> (Signature) .....
>
> (Place of abode).....

Copies of such certificates may be obtained from the Government Printer, the Reception House, Darlinghurst, or at the above address.

A further amendment to the Act also makes legal the voluntary admission of patients to a mental hospital. The Department has for some time admitted patients in this manner, but there was previously no legal sanction for the Department's action.

The provision in this regard, to which attention is specially called, is as under:

> 24A. (1) The superintendent of any hospital for the insane or licensed house may receive into such hospital or licensed house as a voluntary patient any person who is desirous of submitting himself for treatment.
>
> No person shall be so received unless with the assent in writing of the Inspector-General, who shall only give such assent upon the written application of the person desiring to become a voluntary patient.
>
> (2) Every such patient shall be produced to the Inspector-General at his visits.
>
> (3) No such person shall be detained for more than seven days after having given notice to the superintendent of his intention or desire to leave.
>
> (4) No voluntary patient shall be deemed to be an insane patient or a patient within the meaning of section three. The Master may, but only at the request in writing of a voluntary patient, take charge of his property, and the Master shall then have in respect thereof the same powers as he has over the property of an insane patient, and unless released by the Master in the prescribed manner, such property shall remain under his care until the voluntary patient is discharged or dies.

(5) The Master may take the necessary steps to collect the cost of maintenance of a voluntary patient from the patient himself, or from the relatives of such patient. For that purpose the provisions of sections one hundred and forty-two to one hundred and forty-seven, inclusive, applicable to or with regard to insane patients and their relatives, shall apply to and with regard to such voluntary patient and his relatives.

(6) Notice of admission and of the discharge or death of such a patient shall be given as in the case of an insane patient.

Yours, etc.,

C. A. Hoog,

Inspector-General of Mental Hospitals,

New South Wales.

January 7, 1935.

Books Received.

DISEASES OF CHILDREN; Third Edition, with contributions by 36 authors, edited by H. Thursfield, D.M., M.A., F.R.C.P., and D. Paterson, M.D., F.R.C.P.; 1934. London: Edward Arnold. Royal 8vo., pp. 1163, with illustrations. Price: 50s. net.

SEX ETHICS: THE PRINCIPLES AND PRACTICE OF CONTRACEPTION, ABORTION AND STERILIZATION, by J. Ellison, B.A., M.B., B.Ch., F.R.C.S., F.C.O.G., A. Goodwin, M.D., B.S., F.R.C.S., C. D. Read, M.B., Ch.B., F.R.C.S., F.R.A.C.S., M.C.O.G., and L. C. Rivett, M.A., M.C., F.R.C.S., M.C.O.G.; 1934. London: Baillière, Tindall and Cox. Super royal 8vo. pp. 232, with illustrations. Price: 12s. 6d. net.

Diary for the Month.

FEB. 1.—Queensland Branch, B.M.A.: Branch.
FEB. 5.—Tasmanian Branch, B.M.A.: Council.
FEB. 5.—New South Wales Branch, B.M.A.: Organization and Science Committee.
FEB. 6.—Western Australian Branch, B.M.A.: Council.
FEB. 6.—Victorian Branch, B.M.A.: Branch.
FEB. 7.—South Australian Branch, B.M.A.: Council.
FEB. 8.—Queensland Branch, B.M.A.: Council.
FEB. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 12.—Tasmanian Branch, B.M.A.: Branch.
FEB. 19.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 19.—Tasmanian Branch, B.M.A.: Council.
FEB. 22.—Queensland Branch, B.M.A.: Council.
FEB. 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 27.—Victorian Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xiv, xv, xvii.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Honorary Assistant Surgeons.

CHILDREN'S HOSPITAL, CARLTON, VICTORIA: Assistant Resident Medical Officer.

DEPARTMENT OF PUBLIC HEALTH, WESTERN AUSTRALIA: Junior Resident Medical Officer.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer.

LEWISHAM HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Physician.

MOOROOPNA HOSPITAL, MOOROOPNA, VICTORIA: Junior Resident Medical Officer.

RENWICK HOSPITAL FOR INFANTS, SYDNEY, NEW SOUTH WALES: Resident Medical Officer.

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Officers.

ROYAL AUSTRALIAN NAVY: Medical Officers.

THE PRINCE HENRY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Physician.

THE VICTORIAN EYE AND EAR HOSPITAL, MELBOURNE, VICTORIA: Resident Surgeons, Post-Graduates.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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